

**A DESCRIPTIVE STUDY ON THE PREVALENCE OF LOW
BONE DENSITY AND ASSOCIATED MODIFIABLE RISK
FACTORS AMONG INDIVIDUALS AGED 18 YEARS AND
ABOVE IN NANGANALLUR, AN URBAN AREA OF
CHENNAI IN 2012**

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CERTIFICATE

This is to certify that the dissertation titled **“A DESCRIPTIVE STUDY ON THE PREVALENCE OF LOW BONE DENSITY AND ASSOCIATED MODIFIABLE RISK FACTORS AMONG INDIVIDUALS AGED 18 YEARS AND ABOVE IN NANGANALLUR, AN URBAN AREA OF CHENNAI IN 2012”** is a bonafide work carried out by **Dr. PRIYADARSHINI. C**, Post Graduate student in the Institute of Community Medicine, Madras Medical College, Chennai -3 under my supervision and guidance towards partial fulfillment of the requirements for the degree of M.D. Branch XV Community Medicine and is being submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai.

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ABBREVIATION

BMD	Bone mineral density
BMI	Body mass index
BMP	Bone morphogenetic proteins
BRU	Bone remodelling unit
CI	Confidence interval
CRP	C reactive protein
DALYs	Disability adjusted life years
DEXA	Dual energy x-ray absorptiometry
df	Degrees of freedom
DPA	Dual photon absorptiometry
HRT	Hormone replacement therapy
IL	Interleukin
OR	Odd's ratio
PPI	Proton pump inhibitor
QUS	Quantitative ultrasound
QCT	Quantitative computed tomography
RANKL	Receptor activator for nuclear factor κ B ligand
SD	Standard Deviation
SE	Standard error
TGF	Transforming growth factor
TNF	Tumor necrosis factor
TSH	Thyroid stimulating hormone
USD	US dollars
WHO	World health organisation

INTRODUCTION

1. INTRODUCTION

Low Bone Density is a term which comprises the two entities of osteopenia and osteoporosis. These form a continuum in the disease spectrum where osteopenia refers to milder bone density loss and osteoporosis to severe bone density loss. Osteoporosis has been defined as “a systemic skeletal disease characterised by low bone mass and microarchitectural deterioration of bone tissue with consequent increase in bone fragility and susceptibility to fracture”.¹

The World Health Organisation (WHO) defines the same as Bone Mineral Density (BMD) more than 2.5 standard deviation below the peak BMD of a young reference population. Definition of severe osteoporosis is BMD more than 2.5 standard deviation below the peak BMD and the presence of fractures. Osteopenia is the BMD that lies between 1 and 2.5 SD below the mean value of a young adult.²

Osteoporosis is termed as a “silent disease” and a “silent global epidemic”. It is called a silent disease as bone density is gradually lost over time without manifesting any signs or symptoms until a bone densitometry test reveals bone loss or a fracture occurs with minor trauma. It is called as a silent epidemic as the disease has been rising in proportions without being duly recognised by the medical fraternity and without awareness among the general population.²

osteoporotic patients. An estimated 25 million people are affected by osteoporosis alone in India in 2003 and this is expected to rise to 36 million by 2013.⁵

The direct cost of treating hip fractures in government hospitals across the country surgically is around 150 USD or approximately 10,000 rupees whereas the same at a private hospital is 2500 – 3000 USD or 1,50,000 to 2,00,000 rupees approximately. This places a heavy burden on the people seeking treatment as majority of the population have to spend out of pocket and are not covered by insurance and this does not even take into account the indirect costs incurred.⁴

1.1 Global Scenario

Osteoporosis afflicts an estimated 200 million people worldwide. An estimated 75 million people are affected in the United States, Europe and Japan. Fractures due to osteoporosis are more than 8.9 million annually worldwide. The lifetime risk of an osteoporotic fracture in developed countries is 30 – 40% which is close to that of coronary heart disease. It accounts annually for 2.8 million disability – adjusted life years (DALYs) in America and Europe that is approximately 1% of the DALYs contributed by non communicable diseases.³

It is predicted that one in every three women and one in five men will suffer from an osteoporotic fracture worldwide. The incidence of hip fracture will increase by 310% in men and by 240% in women by the year 2050.

The belief that osteopenia and osteoporosis are diseases of the west is turning out to be a myth with many Asian countries showing a 2 – 3 fold increase in the incidence of hip fractures over the last 3 decades. More than 50% of the osteoporotic hip fractures are expected to take place in Asia by 2050. There is mounting evidence that epidemics of hip fractures are occurring with the wave of urbanisation through Asia.⁴

1.2 Indian Scenario

With a large population and the growing ageing populace due to the increasing life expectancy, India is home to a large number of osteopenic and

OBJECTIVES

2. OBJECTIVES OF THE STUDY

1. To estimate the prevalence of low bone density among individuals aged 18 years and above in Nanganallur, an urban area of Chennai in 2012.
2. To estimate the prevalence of associated modifiable risk factors among the study population.

JUSTIFICATION

3. JUSTIFICATION

1. WHO terms osteoporosis as a ‘silent epidemic’ and as a ‘silent disease’.
2. An estimated 25 million people are affected in India. This shows the high prevalence of the disease.
3. By the time the symptoms manifest the disease is already in an advanced stage. Early diagnosis offers the advantage of lead time where the risk factors of the disease can be modified.
4. Low bone density is attributed to several risk factors of which many are modifiable. This provides scope for modifications of those risk factors identified.
5. Paucity of community based studies on low bone density in India, particularly from Tamil Nadu and especially in the urban areas from among the reviewed literature makes it a necessity to carry out this study.

Hence, this study was undertaken to estimate the prevalence of low bone density and its associated risk factors.

REVIEW OF LITERATURE

4. REVIEW OF LITERATURE

4.1 LOW BONE DENSITY

Low Bone density comprises of two components, osteopenia and osteoporosis. It means a generalised decrease in the bone mass. It is noted that low bone mass and low bone density are terms used interchangeably in the reviewed literature to denote the combined components of osteoporosis and osteopenia.⁶

Osteoporosis was a term coined by Pommer in 1885 to distinguish it from osteomalacia. He defined osteoporosis as a condition with decreased skeletal mass associated with increased porosity. Osteoporosis is characterised by the presence of quantitatively deficient yet qualitatively normal bone. The histologic and chemical analysis of the bone indicates it is structurally normal.

Osteoporosis was defined by the 1990 consensus development panel as “a systemic skeletal disease characterised by low bone mass and microarchitectural deterioration of the bone tissue with consequent increase in bone fragility and susceptibility to fractures”.¹

The World Health Organisation defines osteopenia and osteoporosis as follows:

Table 1: WHO definitions of low bone density based on BMD measurements⁷

Definition	Bone Mass Density Measurement	T-Score
Normal	BMD within 1 SD of the mean bone density for young reference population	T-score more than or equal to -1
Osteopenia	BMD 1–2.5 SD below the young reference population mean	T-score between -1 and -2.5
Osteoporosis	BMD ≥ 2.5 SD below the normal for young reference population mean	T-score less than or equal to -2.5
Severe Osteoporosis	BMD ≥ 2.5 SD below the normal mean for young reference population in a patient who has already experienced ≥ 1 fractures	T-score less than or equal to -2.5 (with fragility fracture[s])

4.2 PREVALENCE OF LOW BONE DENSITY

4.2.1 World

The most common metabolic bone disease seen worldwide is osteoporosis. Low Bone density is commonly regarded as a disease of old age and especially the elderly female. Ross PD had noted that by using the WHO cut off criteria approximately 15% of young adults have osteopenia and 0.6% have osteoporosis. In the United States, Ross states that only 1 in 9 women have normal BMD and 1 in 3 have osteoporosis with the rest having osteopenia.⁸

Studies in the Russian Federation estimate that 14 million Russians have osteoporosis and another 20 million have osteopenia. In Poland an estimated 18.5% women older than 55 years suffer from osteoporosis and 40.7% suffer from osteopenia irrespective of their residential status, i.e., urban or rural.⁹

In studies from Egypt it was seen that among postmenopausal women, 53.9% have osteopenia and 24.8% have osteoporosis. Among men, 21.9% are found to be osteoporotic and 26% osteopenic.¹⁰

4.2.2 Asia

The prevalence of osteoporosis among adult Pakistani women in a study conducted by Fatima M was seen to be 12.9% whereas the osteopenic women constituted 43.4% of the 334 women enrolled in the study.¹¹

A study by Li Yin Ming et al among 498 women and 383 men aged 51 years and above, the proportion of women with osteoporosis and osteopenia was 35.9% and 42% respectively. Among the men, the proportion of osteoporosis and osteopenia was 17% and 42.6% respectively. Overall, 77.9% of women and 60.1% of the men were concluded to have low bone mass.¹²

4.2.3 India

A camp based study conducted by Abraham Samuel Babu et al places osteoporosis at 42.2% among the 609 persons screened in a rural area of north Kerala in India and osteopenia at 40.5%. The males and females both showed high prevalence rates with 64.7% of males and 85.2% of females being affected by low bone density.¹³

Studies by Sharma et al among urban women in Jammu noted that 36.79% of them had osteopenia and 20.25% had osteoporosis. Above the age

of 65 years it was seen that all women had low bone density with 50% having osteopenia and the other half having osteoporosis.¹⁴

Prasad D V in his study conducted among the health care professionals of a hospital at Loni observed that among the 264 subjects in the age groups between 21 – 61 years, osteoporosis was evident in 28.03% whereas osteopenia was seen in 31.06% of them. Among the males, it was 23.33% of osteoporosis and 26% of osteopenia and among the females it was 34.12% and 37.2% respectively.¹⁵

Pande KC's study among healthy Indian population showed that 29.9% of women and 24.3% of men had low bone mass. This study was conducted using digital X ray radiogrammetry.¹⁶

4.3 FRACTURES DUE TO LOW BONE DENSITY

4.3.1 Incidence of fractures due to low bone density

One of the main complications of extremely low bone density or osteoporosis is the occurrence of fractures that affect various bones of the body especially in the elderly. The incidence of fractures is alarmingly high and is predicted to rise.

Osteoporotic fractures are occurring one every three seconds globally and over 50 years of age, one in two women and one in five men will suffer a fracture in their lifetime ahead. By 2050, it has been predicted that the worldwide incidence of fractures would be around 6.3 million per year and Asia and Latin America would be harbouring more than half of these.¹⁷

90% of wrist and hip fractures among the elderly in the United States are due to non-violent trauma.⁸

34 million people in the Russian Federation are at high risk for fractures with 7 vertebral fractures occurring every minute in those over 50 years of age and one hip fracture occurring every 5 minutes.⁹

A study by Tuzun et al at Turkey predicts the annual incidence of osteoporotic fractures at 109 per 1, 00,000 men of age more than 50 years and 226/1, 00,000 women of age more than 50 years for the year 2009.¹⁸

Community based studies are lacking in India but based on hospital studies it can be seen that hip fractures are common here as well. In studies conducted in India by Sankaran among 1393 patients of 3 Delhi hospitals it was seen that hip fracture was common among both sexes with the average age of fractures being 60-70 years.¹⁹

A study conducted among expatriate Indians in Singapore showed that women had higher incidence of hip fractures than men. The Indian population also showed higher hip fracture incidence i.e. 128 men and 361 women per 1,00,000 population as compared to Malays but this was lesser than the incidence among the Chinese population. The peak age for the occurrence of fractures in all of these populations was over 70 years.

Extrapolating these results to the current Indian population, it was estimated that the number of hip fractures each year would be more than

4,40,000 with a 3:1 female: male ratio. This criteria puts projections for 2020 at more than 6,00,000 and for 2050 at more than 1 million.²⁰

4.3.2 Socio-economic burden of fractures due to low bone density

One of the main reasons that low bone density must be prevented, detected and treated is the consequent fractures that occur which in turn place a huge socio-economic burden on the public health system and on the affected individuals even in developed countries and more so in a country like India.

In a study conducted at Sweden, the total costs towards the treatment of a hip fracture works out to approximately 10,00,000 Rupees or 12,200 Euros. The study also reports a poor quality of life among those who had sustained hip and wrist fractures. The cost of community caring and social insurance is high following a fracture.²¹

For the people treated at a public hospital in India for a hip fracture, the direct cost borne by the patient for hospital treatment is approximately 10,000 Rupees and for those treated at a private hospital, the costs maybe up to Rupees 2 lakhs. The public health system pays for the remaining costs incurred by the individual towards treatment in a government set up. This is in addition to the burden of lost wages and diminished quality of life of the affected individual and also an extra burden to care for the patient by the other family members.⁴

4.4 THE SKELETAL SYSTEM

4.4.1 Anatomy of the Skeletal System²²

The skeletal system is made of the bony skeleton and the joints including the cartilage. It provides a rigid frame work which protects and supports the soft tissues of the body. It also acts as a system of levers and struts which permit movement of the body through the muscles attached to it.

Bone

The bone is highly vascular mineralised connective tissue which consists of cells and an intercellular matrix within which the most of the cells are embedded. The matrix consists of 1.the organic matrix which is formed mainly of collagen fibres and 2.inorganic salts (which is rich in calcium and phosphates).

i) Macroscopic Anatomy

The living bone is white in colour and may be either dense in texture or honey combed in appearance. The former is the cortical or compact bone found in the cortices of mature bones and it provides strength. The latter is called cancellous, trabecular or spongy bone. It is formed by a latticework of bars and plates or trabeculae and is usually present internally. Cancellous bone provides additional support to the cortex and supports the bone marrow.

Bone forms a reservoir of calcium and phosphate, the content of which is under hormonal and cytokinal control. The proportions of cortical and cancellous bone varies depending on whether it is a long or flat bone.

ii) Microstructure of Bone²³

Histologically, bone consists of extracellular mineralised matrix and a number of various cell types which include osteoclasts, osteoblasts and osteocytes, the cells of its vascular and nervous supply and the components of the periosteum, endosteum and the marrow.

a. Bone matrix

It is the extracellular mineralised material of the bone. It consists of the ground substance in which collagen fibres are embedded in parallel. 10 -20% of the matrix is made of water. The dry weight of the matrix contains 60-70% of inorganic mineral salts (hydroxides, hydroxyapatite, microcrystalline calcium and phosphate), 30-40% of collagen and 5% of non collagenous protein and carbohydrates mainly glycoproteins.

Before mineralisation, the matrix is called as the osteoid. Adults have very less osteoid which reflects the local site remodelling of bone in which mineralisation follows depletion of the organic matrix.

b. Collagen

It is synthesised by the osteoblasts and is mainly type I collagen. It provides mechanical strength to the bone.

c. Non collagenous organic matrix

It is complex and contains various macromolecules in small amounts attached to the collagen fibres and to the surrounding bone crystals.

d. Osteonectin

The osteoblasts secrete this phosphorylated glycoprotein. It binds to collagen and hydroxyapatite and initiates hydroxyapatite crystallisation.

e. Osteocalcin

Osteocalcin is another glycoprotein synthesised by osteoblasts. It binds to calcium and hydroxyapatite and it is a marker of new bone formation. It is required for bone mineralisation.

The matrix also contains proteases, protease inhibitors and many growth factors secreted by the blasts.

f. TGF – β

Osteoblasts and osteoclasts secrete TGF – β . It is activated in acidic conditions of the ruffled border zone of the osteoclast and acts as a coupling factor for stimulating new bone formation at resorption sites.

g. Bone minerals

These form the inorganic constituents of the bone matrix. It provides hardness and rigidity to the bone and it is the main reason for the bone being visible on a radiograph (50% mineralisation of the bone is needed for it to be visible on a X ray). It is largely composed of crystals made of hydroxyapatite $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH}_2)$ and a small amount of calcium and phosphate. The major ions in the matrix are calcium, phosphate, hydroxyl and carbonate. Citrate, sodium, potassium, magnesium, fluoride, chloride, zinc, copper, aluminium, lead, strontium, silica and boron are seen in less numerous quantities.

h. Osteoblasts

Osteoblasts are derived from osteoprogenitor cells and have features of protein secreting cells. They are seen on the forming surfaces of growing or remodelling bone where they constitute a monolayer covering. Osteoblasts are responsible for the synthesis, deposition and the mineralisation of bone matrix which they secrete. Once they are embedded in the matrix they change into osteocytes. The major activity of osteocytes is to synthesise and secrete organic matrix. It secretes other products like osteonectin, osteocalcin, osteopontin, RANKL (Receptor activator for Nuclear factor $\kappa\beta$ ligand), osteoprotegerin and some other proteoglycans and growth factors like BMPs (Bone morphogenetic proteins). RANKL is the cell surface ligand for RANK, an osteoclast progenitor receptor protein.

Osteoblasts play a major role in the mineralisation of the matrix. Alkaline phosphatase on the osteoblast raises the local concentration of calcium and phosphate. During bone deposition, osteoblasts via RANKL binding to RANK on immature clasts regulate the extent of osteoclast activity. In the presence of the parathyroid hormone, osteoblasts secrete osteoprotegerin which has higher affinity to RANKL. This causes the osteoclasts to mature and resorption occurs.

i. Osteocytes

It is the major cell type of the mature bone. They play a role in the maintenance of the bone and the death of the osteocytes leads to resorption of the matrix by osteoclast activity.

j. Osteoclasts

Osteoclasts are responsible for the local removal of bone during growth and subsequently during remodelling. Each osteoclast contains a ruffled border which is the site of local bone resorption. They cause demineralisation by release of protons which creates an acidic environment by the release of lysosomal and non-lysosomal enzymes. The cytokines secreted by the osteoblasts and other cells such as the macrophage stimulates the osteoclasts.

k. Osteons

The mechanical properties of bone depend on the type of organisation of the matrix. The organisation may either be of the woven or lamellar type. Woven bone has collagen and fibres irregularly attached and are commonly seen in the fetal bones. They are seen in adults during excessive rapid bone remodelling and repair of fractures. Lamellar bone is seen as lamellae arranged around neurovascular channels as concentric cylinders (Haversian canals). They are found in the adult skeleton.

4.4.2 Physiology of Normal Bone

i) Bone Growth and Modelling

During life, bone undergoes radial and longitudinal growth, modelling and remodelling. During the growth and development in childhood and adolescence, both radial and longitudinal growth occurs. At the growth plates, longitudinal growth occurs. Here cartilage proliferates in the metaphyseal and

epiphyseal areas of the long bones, before subsequently undergoing mineralization to form new bone.

Modelling is the process by which the overall shape of bones change in response to the physiologic influences and mechanical forces, leading to the adjustment of the skeleton gradually to forces that it encounters. There may be widening of bones or change in axis by the removal or addition of bone to the appropriate surfaces by the independent action of osteoclasts and osteoblasts in response to biomechanical forces.

ii) Remodelling

The process by which bone is renewed so as to maintain bone strength and homeostasis of minerals is called bone remodelling. It involves the continuous removal of old bone in discrete packets, the replacement of these packets by newly synthesized proteinaceous matrix and the subsequent mineralization of this matrix to form new bone. This process of remodelling resorbs old bone and helps to form new bone so as to prevent any bone micro damage accumulation.

Remodelling of bone begins even before birth and it continues till death. The bone remodeling unit (BRU) comprises of a tightly coupled group of osteoclasts and osteoblasts which sequentially carry out old bone resorption and new bone formation. Perimenopausal and early postmenopausal women show an increase in bone remodelling and then slows with aging but continues at a rate faster than premenopausal women. Aging men have an increased bone remodeling as well. Infants show a 100% turnover of bone in a year whereas in adults the bone turnover rate is 18% a year.

The remodelling cycle comprises of four sequential phases. The four phases in sequence are activation followed by resorption and reversal and finally formation.

a. Activation

This occurs 360 times in an hour in a normal adult. The osteoblasts and haematopoietic precursors initiate osteoclast activation and are sometimes initiated by inflammatory cells, particularly T cells. Osteoblasts initiate activation of osteoclasts by production of RANKL, a ligand for the receptor activator of NF- κ B (RANK) leading to the differentiation of osteoclasts and maintenance of their function. Osteoprotegerin (OPG) also secreted by osteoblasts, is a decoy receptor that can block RANKL/RANK interactions and inhibits the activity of osteoclasts.

b. Resorption

On activation, multinucleated osteoclasts are formed by the fusing of circulating monocytes which degrade the organic and inorganic components of the matrix by their ruffled border. This creates a cylindrical tunnel by its concerted action and a 'cutting cone' is formed by groups of osteoclasts.

c. Reversal

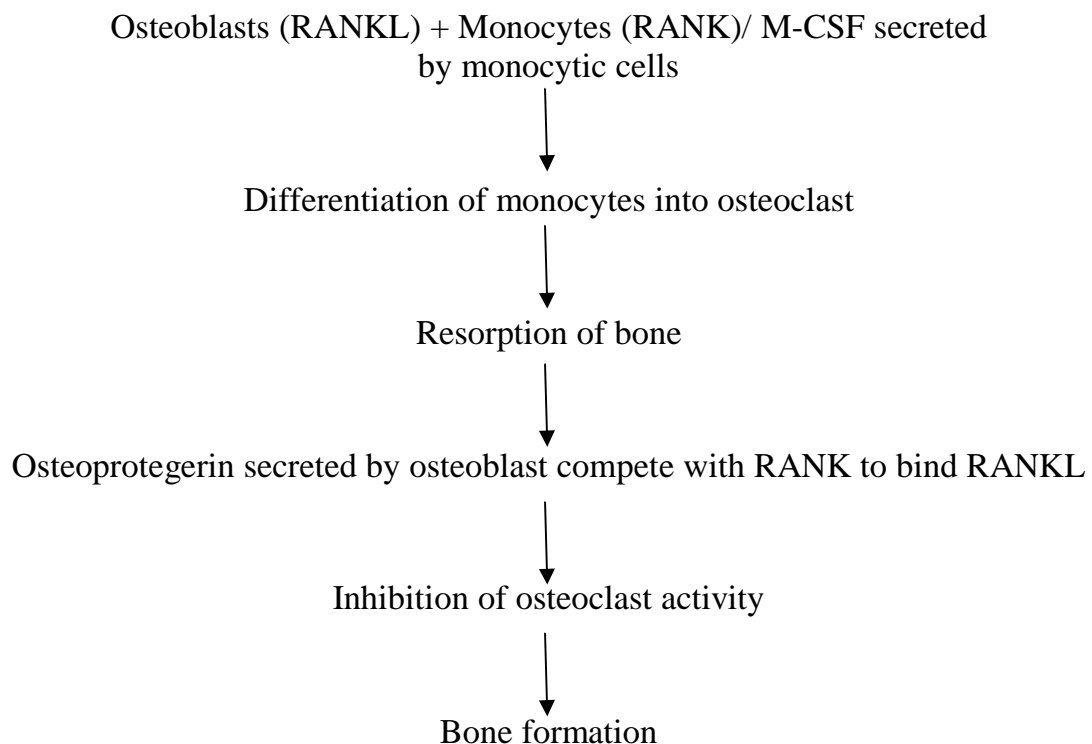
During the reversal phase, the osteoclasts and resorbing cells die by apoptosis and are replaced by osteoblasts. These cells track the osteoclasts as they tunnel through the cortex.

d. Formation

The initiation of the formation phase is possibly by factors produced by the osteoclast or by reversal cells or released from the bone matrix. This phase is quite longer than the first 3 phases and involves progressive waves of osteoblasts producing bone matrix. The osteoblasts fill the space caused by the osteoclasts called the 'closing cone'. These osteoblasts then become flat lining cells and become embedded in the bone as osteocytes or they undergo apoptotic death.

4.4.3 Pathogenesis of bone loss²⁴

Remodelling depends on the balance between the removal and deposition of bone (i.e. balance between the activities of the osteoblasts and that of the osteoclasts). The normal remodeling process may be summarised as follows:



The pathogenesis of bone loss is associated with excess of the above osteoclastic activity i.e. remodelling. Normal humans gain bone in early life and then it plateaus. This is followed by loss of bone as people grow older. When this bone loss is either accelerated or exaggerated, it causes low bone density or osteopenia and osteoporosis.

A recent study among postmenopausal women showed that monoclonal antibodies against RANKL produced prolonged inhibition of bone resorption.²⁵ Another study showed in estrogen deficient early postmenopausal women that on the surface of bone marrow cells, RANKL levels were increased.²⁶

❖ **Role of Estrogen**

Estrogen inhibits the secretion of cytokines such as interleukin-1, IL-6 and Tumor Necrosis Factor- α (TNF- α) which foster the development of osteoclasts and estrogen also stimulates the production of TGF- β which increases the apoptosis of osteoclasts. Adult women normally have lesser bone mass than adult men and after menopause these women have an additional deficiency of estrogen. This causes an increase in the bone loss.

❖ **Calcium, Vitamin D and Parathyroid Hormone**

A clinical trial conducted among older individuals who were highly predisposed to calcium and vitamin D indicated that the supplementation of both can lead to reversal of secondary hyperparathyroidism, reduction in the

bone resorption, increase in the bone mass, reduction of fracture rates and even reduce the frequency of falling. However, in a recent large study it was seen that calcium and vitamin D supplementation did not bring down the fracture incidence significantly, maybe because the studied population was less deficient in vitamin D.²⁷

❖ **Role of prostaglandins and cytokines**

The concept that the cytokines produced locally such as IL-1 and prostaglandins like prostaglandin E₂ (PGE₂) affecting bones is more than 30 years old. Recently, many cytokines have been found to either stimulate or inhibit bone resorption and formation. Both stimulatory and inhibitory actions are displayed by prostaglandins. However, the predominant effect of the major prostaglandin produced by bone cells (PGE₂), is stimulating both resorption and formation. Prostaglandins, especially PGE₂, are produced by bone cells largely by the action of cyclooxygenase 2 (COX2). It is induced by many of the factors that stimulate bone resorption and therefore enhance the response to these agents.²⁸

❖ **Collagen abnormalities**

The first intron of the gene coding for type I collagen $\alpha 1$ chain if showing a particular polymorphism can influence fracture risk independent of BMD along with increased levels of homocysteine.²⁹

4.4.4 Summary of pathogenesis of Low Bone Density

To summarise, the bone remodelling cycle is extremely complex. Osteocytes, osteoblasts and osteoclasts, the three major cells arising from different stem cells (mesenchymal for osteoblasts and osteocytes and hematopoietic for osteoclasts) are controlled by a variety of factors which harmoniously orchestrate a systematic remodeling sequence. The birth and death of these cells along with the regulatory factors which control those events, although complex, are essentially important for understanding the pathogenesis of osteoporosis. Alterations in any stage of the process of recruitment, activation, differentiation or cell death can lead to imbalance in remodelling which would ultimately result in bone loss or reduced bone mass or density.

4.5 RISK FACTORS FOR LOW BONE DENSITY

Low Bone Density may occur as a result of many complex risk factors each contributing to increased osteoclastic activity thereby causing increased bone resorption. The risk factors may either be modifiable or non modifiable.

4.5.1 Modifiable Risk Factors

Low bone density is caused by a myriad of causes working together. There are proven modifiable risk factors which cause low bone density and they include the following:

- Alcohol
- Smoking
- Low body mass index

- Poor nutrition
- Vitamin D deficiency
- Estrogen deficiency
- Caffeine intake
- Insufficient exercise

i) Alcohol

On comparing with individuals who consume alcohol in moderate amounts and those who do not consume any alcohol, people consuming alcohol in excess of 2 units a day have a 40% increased risk of sustaining any osteoporotic fracture. High alcohol intake leads to secondary osteoporosis by its direct effects on the osteoblasts and on the parathyroid hormone that regulates calcium metabolism and by also causing poor nutritional status (protein, calcium and vitamin D deficiency).³⁰

Use of any alcohol was seen to produce lower bone density among adolescents than those who did not in a study by Korkor et al.³¹

A study by Tucker et al among men, premenopausal and postmenopausal women of the Framingham Offspring cohort showed that high intake of liquor (> 2 units per day) was associated with lower bone mineral density.³²

ii) Smoking

People with history of having ever smoked cigarettes and people who currently smoke are at increased risk of fractures as compared to non-smokers.³³

Studies by Szuluc indicated that bone density was low among individuals who either are either current smokers or past smoker.³⁴

iii) Low body weight and body mass index

People with higher values of body mass index also tend to have higher values of BMD. A positive correlation is seen between body mass index and bone mineral density. People who have a BMI less than the normal, i.e. BMI < 18.5 kg/m² are at risk of developing low bone density. Obesity acts as a protective factor in the development of low bone density.³⁵

A community based study conducted in Sri Lanka by Lekamwasam among men aged 50 years and above showed that the individuals in the higher tertile have higher mean body weight than those in the lower two tertiles.³⁶

Funakoshi's study indicates a very significant positive correlation between bone density and weight ($r=0.45$, $p < 0.001$) and BMI ($r=0.48$, $p < 0.001$).³⁷

iv) Poor nutrition

The intake of insufficient calcium in diet or its inefficient absorption leads to the increased production of parathyroid hormone which hikes bone remodelling causing mobilisation of osteoclasts and thereby reducing bone

density. For men and women aged 50 years or less, the recommended intake of dietary calcium is 1,000 mg/day. For those above 50 years, the intake recommended is 1,200 mg/day.³⁸

The bone calcium content shows positive correlation with bone mass. People with BMI <23 kg/m² have lower bone density values than those with BMI >27 kg/m². Overweight or obese women, have better calcium absorption and also lesser post climacteric bone mass loss, than those with normal body weight. Women who have lower BMI have higher bone fracture risk caused by lower bone density and lesser subcutaneous fatty tissue. It is acceptable if older women are to be a little overweight for protection of osteoporosis, as long as risk factors for diseases like hypertension, type 2 diabetes, dyslipidaemia and other cardiovascular diseases are absent³⁹.

A randomised controlled trial conducted among healthy postmenopausal women found that calcium supplementation lowered the rate of loss of bone density among women who had low levels of calcium intake. Women who were put on placebos had greater degrees of bone loss.⁴⁰

A Swedish study demonstrated that those who were in the lowest quintile of calcium intake had higher numbers of fractures and at an increased rate as well. The percentage of individuals who are osteoporotic reduced with the increase in calcium intake.⁴¹

v) Vitamin D deficiency

Vitamin D is essential since it helps in calcium absorption from the intestines into the blood. It is also made in our skin with exposure to the sun's ultraviolet rays. In many people casual exposure to the sun for at least 10-to-15 minutes a day is usually sufficient. However in the elderly, people who do not go outdoors often or do so covered completely, supplemental sources of vitamin D is of much importance. At least 800 international units of vitamin D and 1,000 to 1,500 mg of calcium daily can protect against osteoporosis⁴².

People who took adequate quantities of milk and calcium rich diets were seen to have better bone mineral densities as compared to those who took them in lesser quantities or avoided dairy foods.⁴³

vi) Estrogen deficiency

Estrogen deficiency in women leads to bone loss in a similar fashion that occurs in post menopausal women. It could also worsen the situation if occurring in adolescence and early adulthood by preventing the build up of bone mineral density.

Estrogen supplementation as treatment for low bone density has been well documented to delay the bone loss among post menopausal and in some cases even improve the bone density however, its effects maybe attenuated in the presence of other risk factors for low bone density such as tobacco use and alcohol consumption.⁴⁴

vii) Caffeine intake

Caffeine intake if excessive from sources such as coffee, tea or cola drinks can cause reduced bone density. Caffeine acts by increasing the apoptosis by osteoblasts thereby reducing the bone mineralisation.⁴⁵

A study conducted among middle aged women showed a positive relation between the intake of caffeine and the risk of hip fracture and the relative risk of fractures among women in the highest quintile of caffeine consumption was 2.95.⁴⁶

viii) Insufficient exercise

People who practice a more sedentary lifestyle are more likely to have hip fractures than those who are more active. Studies suggest that post menopausal women who were physically active, i.e at least had moderate physical activity for at least 30 minutes on most days of the week as per the WHO recommendations had lesser incidence of falls and fractures.⁴³

Men above 50 years of age who are less physically active have lower bone densities compared to the men who are highly physically active this being statistically significant ($p= 0.0001$) in the study by Lekamwasam.³⁶

4.5.2 Non-modifiable risk factors:

Apart from the above risk factors which can be modified to prevent low bone density, there are other non modifiable risk factors. The non modifiable risk factors for low bone density are as follows:

- Increasing age
- Gender
- Genetic factors
- Late menarche
- Early menopause or hysterectomy with oophorectomy
- Family history of osteoporosis
- Previous fractures

a) Age

Age plays a major role in thinning of bones and occurrence of low bone density as there is definite decrease in the BMD as age increases. Peak bone mass is attained close to the age of 30 years and is influenced by the presence or absence of various risk factors which affect bone mineral deposition. Thereafter, a reduction in the bone mass occurs and if accelerated could lead to low bone density at younger ages. Hence, the onus is on improving the peak bone mass by increasing bone deposition in the formative years and reducing the rate of bone loss in later years.⁴⁷

A linear age related decline in the bone density is seen among women as they were followed from the ages of 21 – 68 with the attainment of peak bone mass seen at the end of the linear skeletal growth among these women.⁴⁸

A study among south Indian men from the age of 41 onwards shows a significant decline in the BMD of the spine and hip as the age progresses.⁴⁹

b) Gender

Gender plays a distinctive role as a risk factor for low bone density. Women are far more predisposed to low bone density than men at all ages. Women also attain lower peak bone mass than men and also show greater decline in bone density at later ages this being marked postmenopausally. The rates of fracture though are equal for a given BMD for both men and women. The T scores calculated to assess low bone density are based on the particular sex and finally give results which maybe compared between sexes.⁵⁰

c) Genetic factors

Blacks tend to have the highest bone densities among all races. Caucasian individuals tend to have lower BMD than their black counterparts at all ages. Asian and Hispanic people have even lesser BMD than their white counterparts.^{54, 51}

Race also affects estrogen and Vitamin D sensitivity with the Asians being more estrogen resistant than Caucasians but more Vitamin D sensitive.⁵²

d) Late menarche and early menopause

Sex hormone deficiency plays a role in the development of low bone density. Late menarche and early menopause both lead to shorter durations of exposure to the sex hormones among the females. This impacts the deposition and resorption of bone as the protective effect of estrogen is lost.⁵³

Hysterectomy accompanied by oophorectomy also accelerates the rate of loss of bone density among women.⁵⁴

e) Family history of osteoporosis

Family history of osteoporosis is a strong predictor of osteoporosis in an individual. A study conducted among U.S. women, 19.8% of women with osteoporosis had a family history positive for osteoporosis and this was significantly and independently associated (OR = 2.35, 1.87 – 2.96). When more than one relative was affected the association strengthened (OR= 8.48, 4.5 – 15.9).⁵⁵

A study conducted in California showed that the BMD levels among individuals whose family members had positive history of osteoporosis was lower than those who had a negative family history ($p < 0.02$).⁵⁶

f) Previous history of fractures

Sustaining fractures is independently a risk factor for the development of osteoporosis and further fractures. Previous fragility fractures strongly indicate the investigation of bone mineral density.⁵⁷

4.5.3 Secondary causes of low bone density⁵⁸

Low Bone density also occurs due to pathologies which cause increased osteoclastic activities leading to accelerated bone loss and also prevent the normal deposition of bone minerals. These may be classified as below:

Table 2: Secondary Causes of Low Bone Density

I. Endocrine disorders	II. Rheumatologic disorders
Hyperthyroidism	Rheumatoid arthritis
Primary hyperparathyroidism	Juvenile polyarticular arthritis
Type 1 diabetes mellitus	Ankylosing spondylitis
Cushing's syndrome	Systemic lupus erythematosus
Hypogonadism	IV. Malignancy
III. Pharmacotherapy	Multiple myeloma
Excessive glucocorticoids	Leukemia
Anticonvulsants	V. Gastrointestinal disease
Intravenous heparin	Pernicious anemia
Chronic L-thyroxine replacement	Celiac disease
Proton pump inhibitor (PPI) use	Gastrectomy, small/large bowel resection
Antacid over use	Inflammatory bowel disease
VI. Chronic obstructive liver disease	VII. Renal insufficiency or failure
Primary biliary cirrhosis	IX. Genetic diseases
VIII. Immobilization	Marfan's syndrome
Prolonged bed rest or wheelchair-bound	Osteogenesis imperfecta
Space flight	Sickle cell anemia
X. Miscellaneous causes	Thalassemia
Anorexia nervosa or bulimia	Congenital porphyria
Vegetarian diet	Glycogen storage diseases
Lactose intolerance	Homocystinuria
Prolonged parenteral nutrition	Hypophosphatasia
Pregnancy and lactation	Ehlers-Danlos syndrome
XI. Idiopathic causes	Multiple dystrophy
Juvenile osteoporosis	Multiple sclerosis
Idiopathic osteoporosis of young adults	

4.6 SYMPTOMS AND CONSEQUENCES OF LOW BONE DENSITY

Low bone density does not usually cause any symptom. The thinning of bones often goes unnoticed until a fracture occurs. Sometimes, chronic back pain which is aggravated by movements may occur as a result of osteoporosis.⁸

4.6.1 Osteoporotic Fractures

Mild or moderate trauma that does not affect normal young healthy adults is sufficient to cause severe fractures among the osteoporotic individuals especially the elderly. Falls from a standing height or lower are sufficient to cause serious hip or wrist fractures among them.

Fractures due to low bone density most commonly involve the following: wrists, femoral neck and vertebral bodies. The metatarsals, humerus, ribs, toes, leg, pelvis, hand and clavicle are also commonly fractured due to low bone density.⁵⁹

4.7 DIAGNOSIS OF LOW BONE DENSITY

4.7.1 Bone density tests

There are various tests to measure bone density and to diagnose low bone mass. The tests commonly used in clinical practice are:

❖ DEXA (Dual Energy X-ray Absorptiometry)^{60, 61}

Dual energy x-ray absorptiometry (DEXA) otherwise known as bone densitometry or bone density scanning, an enhanced form of x-ray technology

is used to measure the bone density. It is the most accurate method of measuring BMD in all the available methods. It makes use of beams of X-rays which estimate the hip and spine bone density. X-ray beams pass easily through weak bones which are less dense. Strong and dense bones allow less X-rays to pass through them. The amount of X-rays which are blocked by bone and soft tissue are compared to each other. DEXA can measure as little as 2% of bone density loss in a year. The DEXA test results are given in the form of two scores:

T score — The T score value gives the amount of bone a person has compared to a young adult of the same gender with peak bone mass. This score can be used to estimate the risk of developing a fracture.

Z score — The Z score value reflects the amount of bone a person has compared with other people of the same age group and gender. If this score is unusually low, it indicates the need for further medical tests.

Benefits

- DEXA is a procedure that is simple, quick and non-invasive.
- The radiation from DEXA is extremely small, i.e. one-tenth the radiation dose of a chest X-ray.
- Of the available methods for bone density testing, DEXA is the most accurate method available at present for diagnosing low bone density and it is also considered as an accurate estimator for fracture risk.

Limitations

- The risk of radiation however small is present in DEXA.
- Pregnant women cannot undergo DEXA.
- Expensive
- DEXA tests are not helpful in following response to treatment.

❖ QUS (Quantitative Ultrasound)⁶²

In the past, DEXA which is a relatively costly and time consuming procedure has only been used for screening of osteoporosis. However, now it is possible to measure bone density with a small and portable ultrasound unit, the quantitative ultrasound machine designed exclusively for bone density testing. QUS measures the heel calcaneal bone density. The hip and spine density measurements from the DEXA are comparable to the heel measurements derived by QUS. QUS measures the speed of sound and broadband ultrasound attenuation using high frequency sound waves. Therefore it analyses the bone density by measuring both how fast sound travels through the bone, and by how much of that sound reaches the other side. The results of the QUS are also given in terms of the T score and are interpreted like the DEXA T score results.

Benefits

- The procedure is completely non-invasive and painless.
- It does not involve the use of radiation
- Inexpensive (lower cost than DEXA)

- Relatively simple to implement and process
- Portable

Limitations

- QUS has lesser precision than DEXA.
- Compared to DEXA, QUS has limited usefulness for monitoring and comparing the effect of medications used to treat osteoporosis.
- Subjects classified as osteoporotic using this method require further investigation such as DEXA to confirm the diagnosis.

❖ Quantitative Computed Tomography (QCT)

Quantitative Computed Tomography can only be done in certain laboratories and needs to be done by following strict protocols. QCT is a type of CT scan that measures the spine bone density. Peripheral QCT (pQCT), a form of QCT measures the density of bones in the arms or legs, usually the wrist. The T scores obtained from QCT are lower than those measured by DEXA because the QCT measurements decrease more rapidly.

Benefits

- QCT can measure trabecular and cortical bone separately which is advantageous to monitor treatment.
- It is the only available modality which directly measures the volume of bone, which can be expressed directly as density.
- Can be performed on a standard hospital CT.

Limitations

- The reproducibility of QCT is poor in community settings
- Less accurate than DEXA or dual photon absorptiometry
- Relatively high radiation dose involved
- Expensive

❖ Dual photon absorptiometry (DPA)

DPA uses a radioactive substance to measure the density of bone. It can measure BMD in the spine and hip. It also uses very low doses of radiation but scans much slowly than the other methods. ^{153}Gd is a common radionuclide used which provides dual energy peaks at 44 & 100keV photons which were counted separately by scintillation detectors. The accuracy of photon absorptiometry is affected by inhomogeneity of soft.

Limitations

- Radionuclide decays and has to be replaced regularly
- Long scanning times due to low photon flux (~40 mins)
- Poor spatial resolution.

4.7.2 Other investigations

For those diagnosed with osteoporosis by one of the above methods, an initial screening laboratory profile should be done. The secondary causes of

osteoporosis need to be done on an individual basis. Further recommended investigations for those diagnosed with osteoporosis are as follows:

i) 25 hydroxy vitamin D level

The optimal level of 25 hydroxy vitamin D in blood is ≥ 30 ng/mL to maximally suppress parathyroid hormone secretion. It is important to have it measured prior to the initiation of any pharmacologic treatment for osteoporosis so as to ensure that the stores of vitamin D are adequate before therapy.

ii) Serum calcium

Measurement of serum calcium enables to rule out the presence of hypocalcemia (malabsorption/vitamin D deficiency) or hypercalcemia (hyperparathyroidism). This is needed to correct the presence of hypocalcemia before initiating pharmacologic therapy for osteoporosis.

iii) 24-hour urine calcium excretion

This enables to diagnose malabsorption (e.g. celiac sprue) and vitamin D deficiency where its value is low. It is high in hypercalciuria, a correctable cause of bone loss.

iv) Serum creatinine

Serum creatinine should be drawn in order to calculate the 24-hour urine calcium excretion and in order to ensure safety of newer pharmacologic osteoporosis therapies.

v) Thyroid stimulating hormone (TSH)

TSH should be measured in patients who are on thyroid hormone supplementation.

4.7.3 Investigations for secondary causes of osteoporosis

The other more extensive evaluation for secondary causes of osteoporosis needs to be considered as indicated on an individual basis. This evaluation may include the following tests:

Table 3: Investigations for secondary osteoporosis

Alkaline phosphatase	Liver function tests
Serum Phosphorus	Complete blood count
Erythrocyte sedimentation rate / C-reactive protein (CRP)	Reproductive Hormones - Lutenising hormone , Follicle stimulating hormone, testosterone, estradiol
Tissue transglutaminase	24-hour urinary free cortisol
Serum and urine protein electrophoresis	

4.7.4 WHO fracture risk assessment tool (FRAX) questionnaire

In addition to the above investigations, it is necessary to administer the FRAX questionnaire to the individuals to assess the 10 year probability of sustaining a fracture. It was developed by WHO and has questions on 10 independent risk factors of osteoporotic fractures. It has 12 questions in all on age, sex, height, weight, history of previous fracture, fracture in parents, presence of rheumatoid arthritis, secondary osteoporosis, glucocorticoid use, alcohol intake, smoking and the bone mineral density.⁶³

4.8 MANAGEMENT OF LOW BONE DENSITY

All patients with low bone density need not be started on pharmacologic treatment. People with osteopenia are advised non pharmacologic interventions to prevent progression to osteoporosis whereas people with osteoporosis and at high risk of fractures are treated with medications.

4.8.1 Non – Pharmacologic management

For patients with low bone density but at low risk of fractures, the following recommendations on life style modification need to be given:

➤ Body habitus

A healthy body weight must be maintained (BMI between 20 -25 kg/m²). This forms an important intervention for the primary prevention and management of low bone density in the earlier stages. Counselling about the same must be given.

➤ Diet

A balanced diet consisting of dairy products and other foods rich in calcium should be advised to the patients. Those with low bone density must be advised to consume a minimum of three portions of milk and dairy everyday and fish eating on at least one day a week.³⁹

➤ Calcium

Adequate calcium from food sources and supplements promote the health of the bones. When dietary sources are not providing enough calcium, supplements may be used to meet this goal.

Daily elemental calcium recommended for healthy individuals from diet and supplement is 1,000 mg for those of 19-50 years and 1,200 -1,500 mg for those over 50 years.

➤ **Vitamin D**

Adequate vitamin D must be taken to support calcium absorption and bone metabolism. Since there is evidence that even abundant sunlight exposure cannot be assumed to produce the much needed vitamin D, dietary and supplementary sources are essential.

➤ **Exercise**

Exercise contributes to many short term as well as long term benefits. People with low bone density must be encouraged to take up exercises for a lifetime that they will continue to practice and enjoy. Muscle strengthening exercises are seen to be an integral part of prevention and treatment of osteoporosis. Exercise produces positive effects in individuals of all ages and regular physical activity in early life helps to achieve higher levels of peak bone density.

➤ **Smoking cessation**

Smoking cessation counselling must be done at every visit to a patient with low bone density. Advice on nicotine replacement therapy must be given.

➤ **Alcohol restriction**

Alcohol use must be restricted to no more than two drinks per day. This limited intake will help to protect the bones and reduce the risk of falls.

➤ **Prevention of Falls**

The vast majority of hip fractures occur after a fall. Preventing falls is a major strategy in the reduction of fracture risk. Modification of the environment, personal risk and medication-associated factors can be effective in preventing falls. In some studies, hip protector pads for frail elderly adults in addition to vitamin D supplementation has been shown to reduce hip fractures.⁶⁴

Treatment for those with osteoporosis and high risk of fracture is with the following therapies:

4.8.2 Pharmacologic Treatment

The goals of treating low bone density with medications are:

1. Reducing the risk of fractures
2. Decreasing bone loss
3. Preserving or increasing bone strength
4. Restoring bone turnover to premenopausal levels in postmenopausal women

For those who have low bone density and are at high risk of sustaining fractures, pharmacologic treatment needs to be considered as given in table 4.

Table 4: Pharmacologic treatment of Osteoporosis⁶⁵

Drug	Mechanism of Action	Indication	Adverse effect
Bisphosphonates Alendronate (Oral) Etidronate (Oral) Risedronate (Oral) Zoledronic Acid (Injection)	Bind to the surfaces of the bones and slows down the resorping action of the osteoclasts. It allows the more effective work by osteoblasts. Thereby, they increase the density of bone and reduce the risk of fractures of the vertebra.	-postmenopausal women with severe osteoporosis -men with severe osteoporosis -men and women of any age who are under chronic steroid medications at risk of fracture	abdominal pain, dyspepsia, nausea, esophagitis, esophageal ulcers, joint/muscle pain, ocular inflammation, osteonecrosis of the jaw, atypical femoral fractures
Synthetic Parathyroid Hormone Teriparatide (sub-cutaneous injection)	works on the bone remodelling process so that new bone is generated and added faster to the skeleton than the old bone being broken down. This is brought about by activating the osteoblasts.	-postmenopausal women with severe osteoporosis - men with hypogonadal (low testosterone) or primary severe osteoporosis -persistent and chronic systemic glucocorticoid therapy in men and women with high fracture risk	nausea, dizziness, leg cramps, transient hypercalcemia, syncope
SERMs (Selective Estrogen Receptor Modulators) Raloxifene (oral)	Act by mimicking action of Estrogen on the bones	- osteoporosis in postmenopausal women	Contraindications: pregnancy, history of venous thromboembolic events Adverse drug effects: flushing, vasomotor

Drug	Mechanism of Action	Indication	Adverse effect
			symptoms, thromboembolic events, leg cramps, flu syndrome
Hormone Replacement Therapy (HRT) Estrogen/ Estrogen + Progesterone (oral/patch)	HRT supplements hormones that are reduced following menopause to the lowest level required to prevent loss of bone. Either estrogen alone or estrogen and progesterone in combination may be given.	-osteoporosis in postmenopausal women who also experience menopausal symptoms like night sweats and hot flashes.	Contraindications: history of thromboembolic events, breast cancer Adverse drug effects: nausea, vomiting, abdominal discomfort, thromboembolic events, breast tenderness, breast cancer, skin irritation on topical application
Calcitonin (Nasal spray)	slows down the work of the osteoclasts and hence allows the osteoblasts to work more effectively.	-osteoporosis in postmenopausal women - osteoporosis in non-pregnant premenopausal women -osteoporosis in men	localized, transient nasal reactions
RANK Ligand Inhibitor Denosumab (sub-cutaneous injection)	anti-resorptive therapy that inhibits the development and activation of osteoclasts	postmenopausal women with osteoporosis	Contraindications: hypocalcemia Adverse drug effects: dermatitis, rashes, cellulitis eczema, pancreatitis, osteonecrosis of the jaw

METHODS AND MATERIALS

5. METHODS AND MATERIALS

5.1 Study design

This study was conducted at the community level as a cross – sectional study to estimate the prevalence of low bone density and associated modifiable risk factors among individuals above 18 years of age at Nanganallur, an urban area of Chennai.

5.2 Study area

The community based study was conducted at Nanganallur of Chennai Metropolitan Area. The area has a residing population of approximately 86,000. The number of residents above the age of 18 years is around 42,100.

5.3 Study duration

The study was carried out from January 2012 to December 2012. The period of field study was from July 2012 to September 2012.

5.4 Study population

The study population comprised of males and females aged 18 years and above in the Nanganallur area. The reason for choosing this population was that though low bone density i.e., osteopenia and osteoporosis are seen as diseases of the aged, the risk factors for low bone density are established at a much earlier age. Individuals within the age group of 18 – 30 years form the most effective group where interventions for modification of risk factors can be taken up as peak bone density is formed at around 30 years of age.

Inclusion criteria

Males and females of age 18 years and above at Nanganallur who were willing to participate were included as part of the study.

Exclusion criteria

- i) Those individuals who are unwilling to participate in the study were excluded and
- ii) Those who are bed-ridden are also excluded as they need to be mobilized for having their heel bone density measured by the quantitative ultrasound machine.

5.5 Sample size

Due to the lack of previous community based studies done among individuals of this age group in the reviewed literature, the sample size was calculated by assuming 50% prevalence of low bone density.

At 95% C.I., $Z_{\alpha} = 1.96$

$p = 50\%$, $q = 50\%$.

d (allowable error, 14% of 50%) = 7 and

Design effect = 2,

$$\text{Sample size} = \frac{1.96 \times 1.96 \times 50 \times 50 \times 2}{7 \times 7} = 392$$

Final sample size was worked out to be 405 as 27 clusters were selected each consisting of 15 individuals.

5.6 Sampling method

The study was carried out by a two stage sampling method. The first stage employed a cluster sampling technique followed by the simple random sampling within each cluster. The electoral list of Nanganallur of Chennai Corporation area was obtained. Each street was considered as a cluster and the total number of clusters in Nanganallur was 278. 15 individuals were chosen from each cluster.

Therefore, total number of clusters = 278

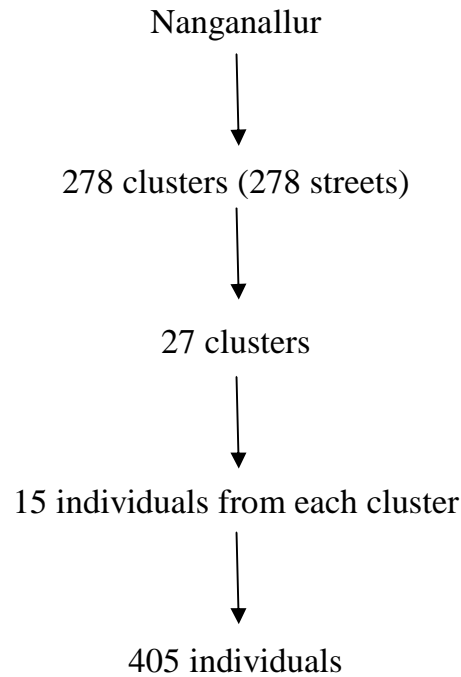
Number of individuals picked from each cluster = 15

Number of clusters picked from the total

$$\begin{aligned} \text{number of clusters} &= \frac{\text{Sample size}}{\text{No. of individuals in a cluster}} \\ &= 392/15 \\ &= 26.133 \text{ clusters} \end{aligned}$$

Therefore, 27 clusters of 15 individuals each was picked resulting in a final sample size of 405 individuals of age 18 years and above.

The selected clusters were arranged in order and the numbers of individuals above 18 years in each of the 27 clusters were arranged alphabetically and they were listed from serial number 1 onwards. By employing the simple random sampling technique using computer generated simple random numbers, 15 individuals were selected from each of the 27 clusters.



In the absence of the individuals selected by simple random sampling at the time of the field visit, the next person on the list was included to be part of the study.

5.7 Study Instruments

A semi structured questionnaire, standard weighing scale, stadiometer and a portable quantitative ultrasound machine were used.

5.7.1 Questionnaire

A pre tested semi structured questionnaire adapted from the Osteoporosis risk test developed by the International Osteoporosis Foundation was used.⁶⁶ The questionnaire was translated into Tamil and again back translated to English to ensure that the meaning of the message conveyed did

not vary. The developed questionnaire was pre-tested and modified to suit the local population. The questionnaire comprised of 2 parts,

1. The socio demographic particulars of the individuals and their family and
2. History regarding the modifiable risk factors – This part elicited the i) fracture history in the individual and their family, ii) diet patterns which contribute to low bone density, iii) physical activity patterns, iv) history regarding systemic medical disorders and medications, v) history of alcohol intake and tobacco usage and vi) menstrual history from females.

The questionnaire was pretested among 35 individuals of age 18 years and above of the Nanganallur area and based on the observations, necessary modifications were made. The results from the pre test were not included in the final analysis.

5.7.2 Measurements

Following the administration of the questionnaire, measurements of height, weight and the bone mineral density were made among the participants of the study.

5.7.3 Height measurement

The height was measured using the same portable stadiometer to the nearest centimetre. The individual was asked to stand erect, looking straight ahead and the height measured without wearing any shoes or chappals.

5.7.4 Weight measurement

The weight was measured by the same calibrated portable weighing scales (Belita®). The individuals were measured to the nearest 100 grams while wearing light clothes and without footwear.

5.7.5 Bone density measurement^{67, 68}

The bone densities of individuals were measured using the Quantitative Ultrasound machine. WHO criteria for diagnosis of osteoporosis and osteopenia using T – score obtained from QUS was used.

The Quantitative Ultrasound by means of its two parameters, the speed of sound (SOS) and broadband ultrasound attenuation (BUA) also assesses the bone structure thus providing better correlation with increased fracture risk. All other investigations for low bone density only give a quantitative assessment of the bone density. However, the definition for low bone density also includes the micro architectural deterioration of bone tissue, a qualitative aspect which is also measured by the QUS. The calibrated QUS machine (General Electric's Achilles®) (Annexure VI) was used to measure the heel calcaneal bone density. The same machine was used throughout the study.

The QUS technique has been applied though DEXA is the gold standard technique available among the non invasive diagnostic procedures. This is because DEXA is more expensive and has a risk of ionising radiation

which is absent with QUS. DEXA qualifies as definitive diagnostic test but QUS is more apt as a screening test.

The heel calcaneal bone density measured using the QUS yields similar results as the hip bone density measurements measured using DEXA. The correlation between the bone density measured using QUS and DEXA is as much as 80-90%.

The Achilles device has a high sensitivity of 96% comparable to that of the DEXA. However, the specificity of QUS is only 55%. This could result in a large number of false positives and hence a DEXA would be needed to confirm the diagnosis in whom low bone density was diagnosed by the QUS but it can be a very effective tool in ruling out those who do not have the disease.⁶⁹

The negative predictive value of QUS has also been studied to be as high as 90% but DEXA shows a marginally greater negative predictive value of 94%.⁷⁰

On comparison of all other modalities of diagnosis, the QUS was chosen as the investigative screening tool for low bone density as it offers the advantages of cost, portability and absence of radiation with good precision of fracture risk prediction. It was also seen to be the most ideal to be employed in a community setting cost effectively.

5.8 Data collection and methods

After obtaining permission from the Director, Institute of Community Medicine and the Dean, Madras Medical College and approval of the Institutional Ethics Committee, data was collected from the study area.

Prior to the collection of data, the investigator underwent training under the guidance of a Radiologist on the use of QUS and interpretation of the data obtained and also under a radiology technician on the operation of the QUS along with its calibration.

A pilot study was done among 35 individuals of the Nanganallur area to pretest the questionnaire and necessary modifications were made. The clusters were chosen by cluster sampling and individuals within the cluster were picked by simple random sampling. The individuals were contacted by going to the house of the individual. The informed consent of the participants were obtained and then they were administered the questionnaire in the local language at their homes. The bone mineral density was then measured using the quantitative ultrasound machine by asking the participants to come to the house of one of the participants where the machine was placed.

The study subject's heel had ultrasound jelly applied over it and was then asked to be placed in the machine. The diaphragm of the machine containing water encased the heels on being started and gave the results of the test within a minute. The machine was calibrated once in two days by using GE's Plexiglas phantom.

Following the T score measurement, if osteopenia was detected the participants were counselled about life style modifications. If osteoporosis was identified the participants were referred for further DEXA tests and also advised life style modifications. The study subjects with risk factors were advised about appropriate modification of the same.

5.9 Analysis Plan

The collected data was entered into a MS excel sheet and analysis was done using the Statistical Package for Social Sciences software version 20. The results are expressed as percentages and proportions. Associations have been done using Chi square tests, Fisher's exact test, correlation and regression. A p value of less than 0.05 has been considered to be significant.

5.10 Operational definitions

5.10.1 Socio economic status

The modified Kuppuswamy scale has been used to classify the socio economic status of the population (Annexure IV).⁷¹

5.10.2 Adequate calcium supplementation

The consumption of supplementary calcium at least one once daily for at least the last 1 month was taken as adequate calcium supplementation.

5.10.3 Adequate milk intake

The intake of 2 or more glasses of milk per day was classified as adequate milk intake.

5.10.4 Adequate fish intake

The intake of fish on at least one day of a week was classified as adequate fish intake.

5.10.5 Adequate ragi intake

Ragi consumption on at least one day of a week was classified as adequate ragi intake.

5.10.6 Caffeine units

The total number of caffeine units consumed by an individual was calculated as one cup of tea equaling 0.5 caffeine units and one cup of coffee equaling 1 caffeine unit.⁷²

5.10.7 Adequate sun exposure

Exposure to direct sunlight for at least 15 minutes every day is classified as adequate sun exposure.

5.10.8 Adequate physical activity

Taking up any sort of aerobic exercise like running, jogging, swimming, bicycling, etc for at least 5 days a week for a minimum of 30 minutes on all those days was taken as adequate physical activity.

5.10.9 Body Mass index

The BMI was calculated as weight in kilograms divided by height in m^2 .⁷³

5.10.10 Osteopenia

T scores less than -1 and more than -2.5 by QUS were classified as osteopenia.⁷

5.10.11 Osteoporosis

T scores less than or equal to -2.5 were classified as osteoporosis.

5.10.12 Low Bone Density

Osteopenia and osteoporosis combined were classified as low bone density.

RESULTS

6. RESULTS

The study has been conducted among 405 individuals of Nanganallur and their results have been presented below.

6.1 Sociodemographic Profile of the Participants

Table 5: Socio demographic details of the participants

Socio demographic parameter		Number	Percentage
Age (in years)	18-30	92	22.7
	31-45	151	37.3
	46-60	131	32.3
	> 60	31	7.7
Sex	Male	137	33.8
	Female	268	66.2
Socio Economic status (Modified Kuppuswamy Scale)	Upper	19	4.7
	Upper middle	190	46.9
	Lower middle	99	24.4
	Upper lower	95	23.5
	Lower	2	0.5
Religion	Hindu	313	77.3
	Christian	68	16.8
	Muslim	24	5.9

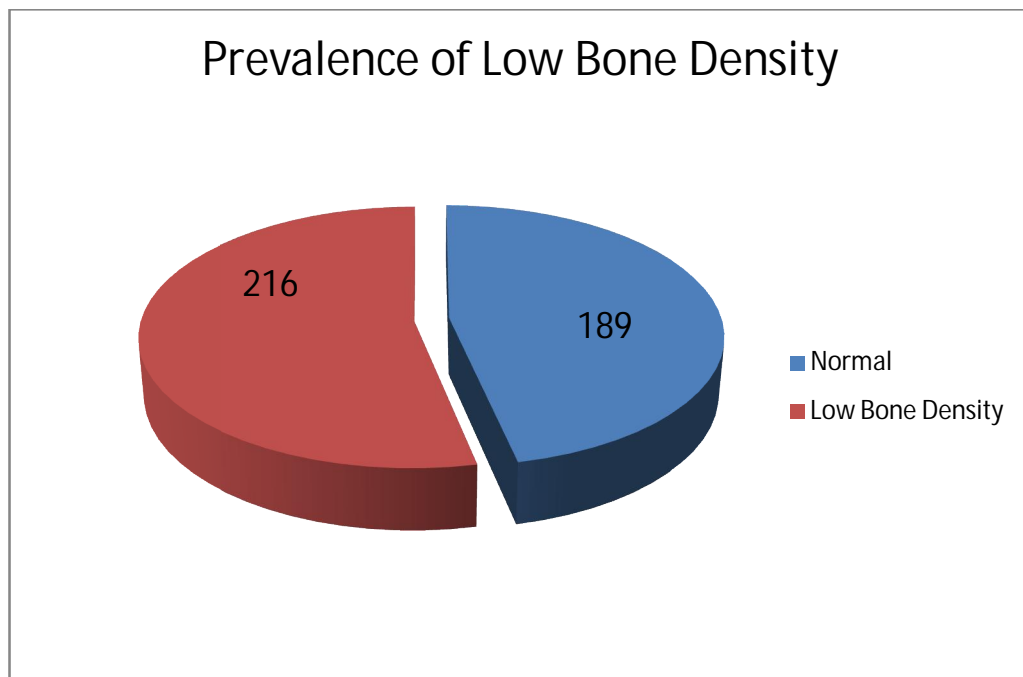
The mean age of the male participants was 45.2 years with a standard deviation of 14.1 years and the mean age of the female participants was 40.23 years with a standard deviation of 12.7 years. The ages varied from a minimum of 18 years to a maximum of 83 years.

6.2 Prevalence of Low Bone Density

Table 6: Prevalence of Osteopenia and Osteoporosis

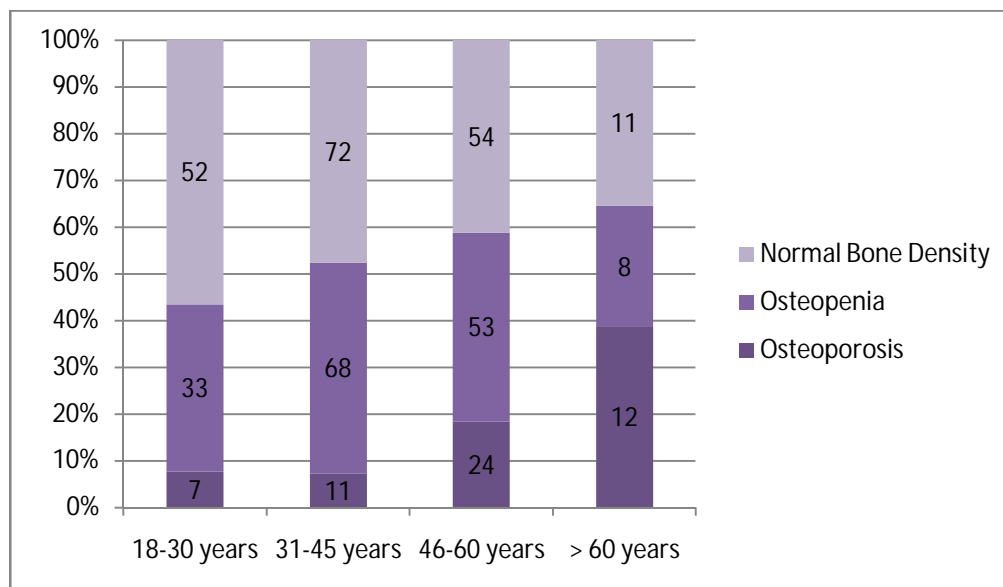
Bone Density	Number	Percentage
Normal	189	46.7
Osteopenia	162	40
Osteoporosis	54	13.3
Total	405	100

Figure 1: Prevalence of low bone density



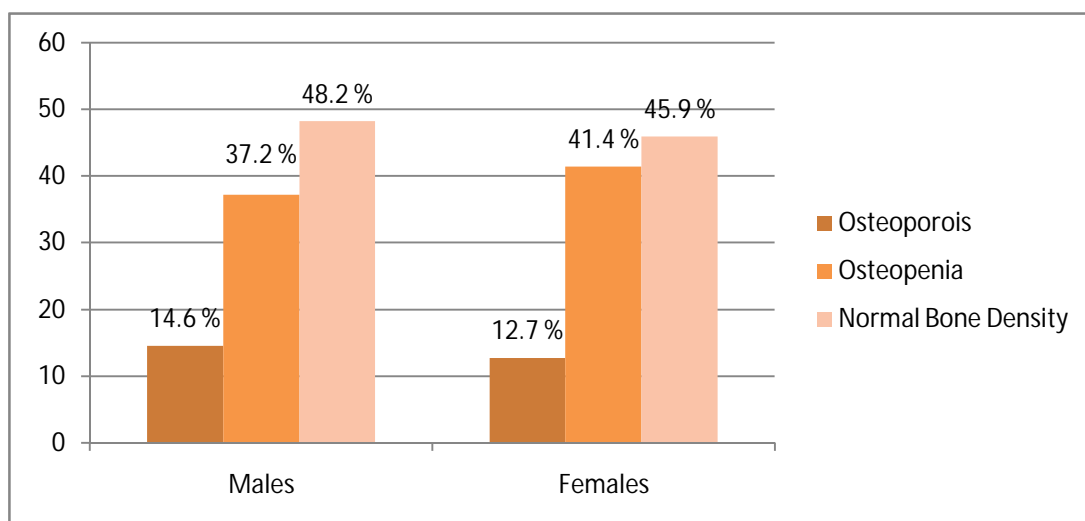
The prevalence of low bone density i.e. osteopenia and osteoporosis combined is 53.3% (Confidence interval – 48.45% - 58.21%).

Figure 2: Age wise prevalence of low bone density



The trend of osteoporosis is seen to increase with increasing age and normal bone density has reduced with increasing age.

Figure 3: Sex wise prevalence of low bone density



The proportion of osteoporotics (14.6%) is higher among males whereas the proportion of osteopenics is higher among females (41.4%).

Figure 4: Religion wise prevalence of low bone density

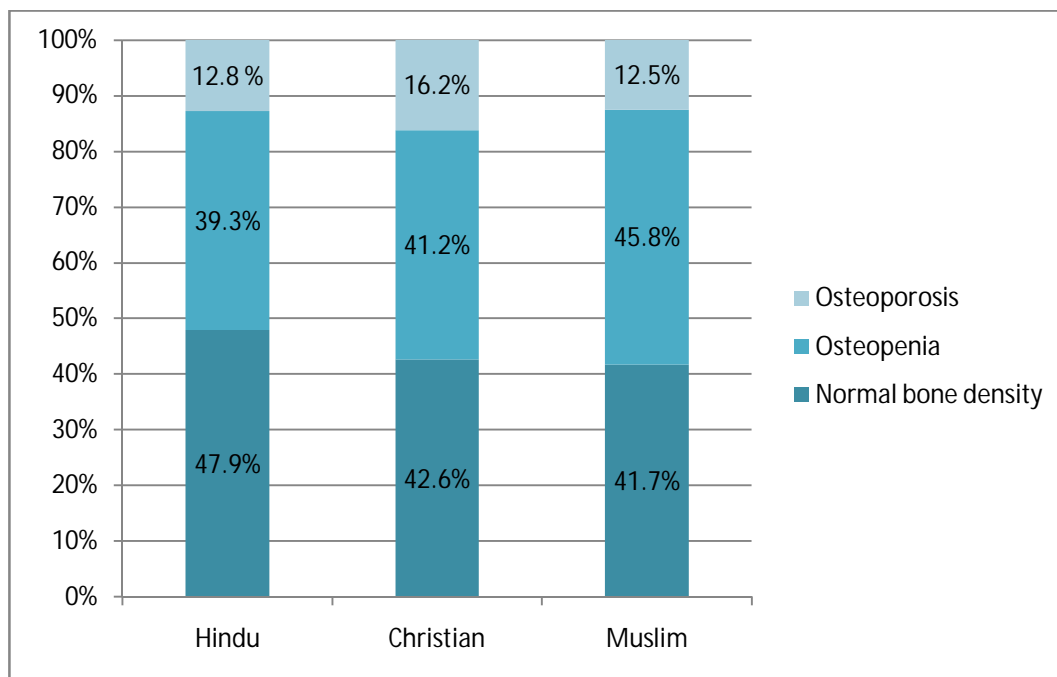
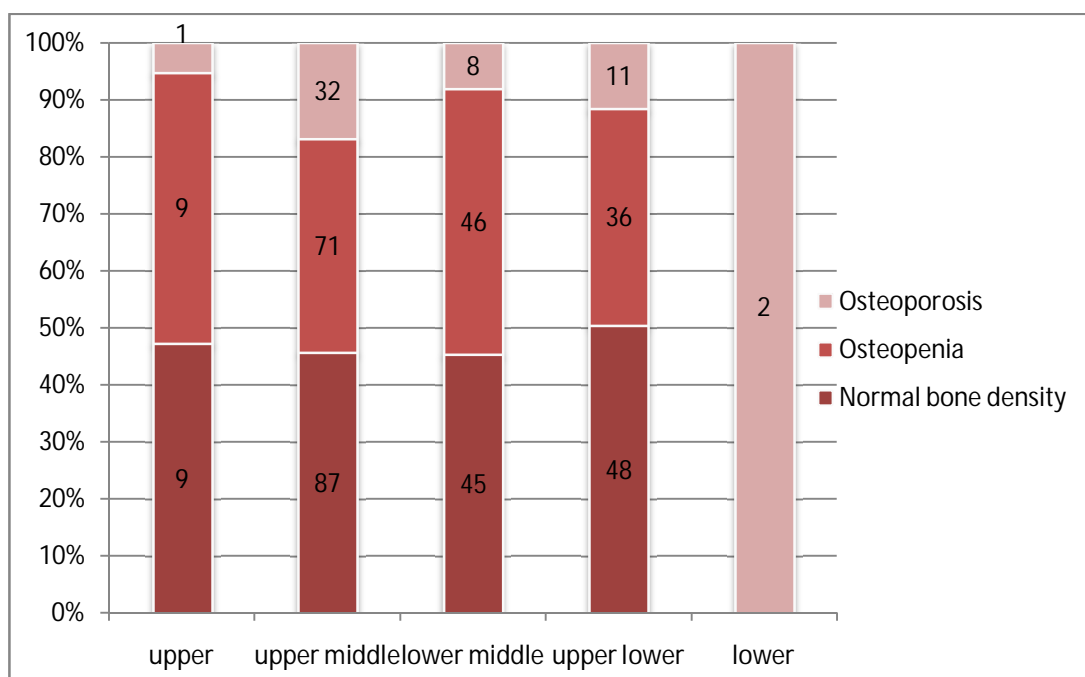


Figure 5: Socio economic class wise prevalence of low bone density



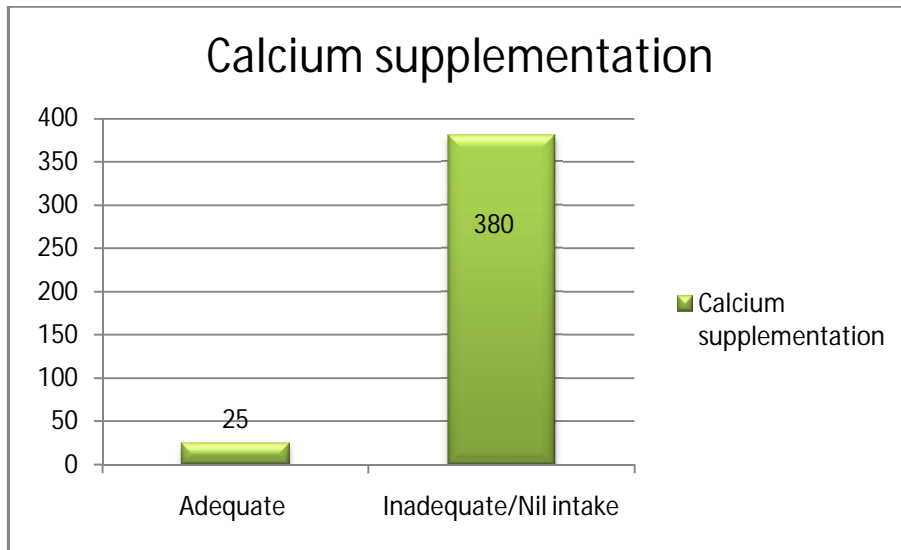
6.3 Prevalence of low bone density risk factors

Table 7: Prevalence of modifiable dietary risk factors

Factor		Number	Percentage
Calcium supplementation	Yes	37	9.1
	No	368	90.9
Fish	Yes	312	77
	No	93	23
Ragi	Yes	131	32.3
	No	274	67.7
Milk	Yes	183	45.18
	No	222	54.81
Coffee consumption	Yes	244	60.2
	No	161	39.8
Tea consumption	Yes	251	62
	No	154	38

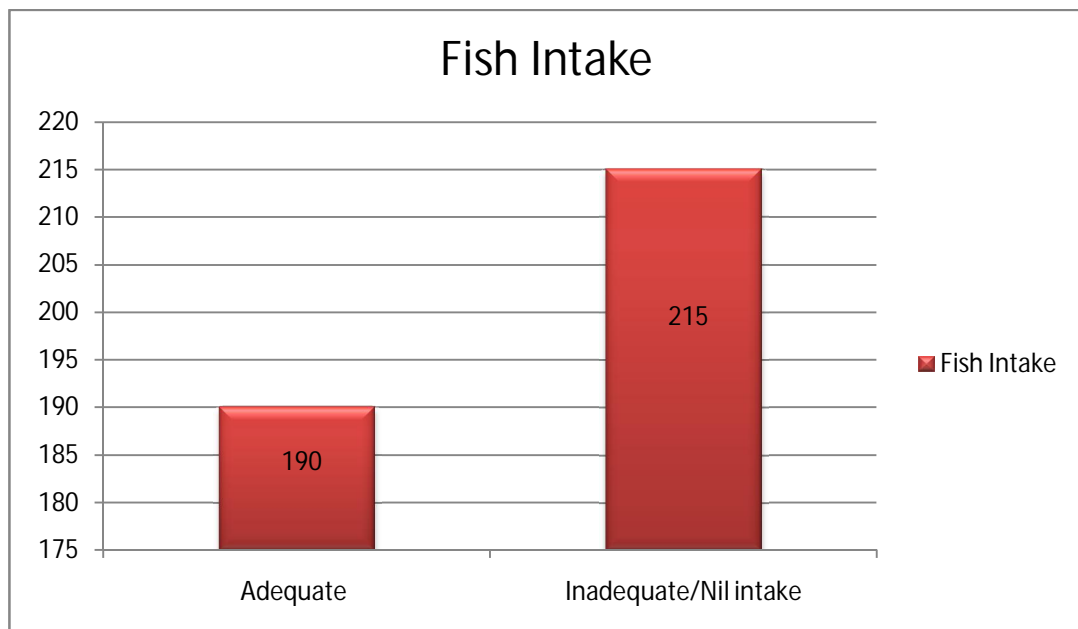
Only 9.1% of the population had regular calcium supplementation. 23% of the population did not consume any fish and 67.7% of the subjects do not consume any ragi at all. 54.81% of the participants avoid milk. Coffee and tea are consumed by 60.2% and 62% of the subjects respectively.

Figure 6: Prevalence of inadequate calcium supplementation



Only 25 (67.5%) of the 37 participants taking calcium are having it in adequate amounts.

Figure 7: Prevalence of inadequate fish intake



46.91% of the participants are having fish once or more in a week.

Figure 8: Prevalence of inadequate ragi intake

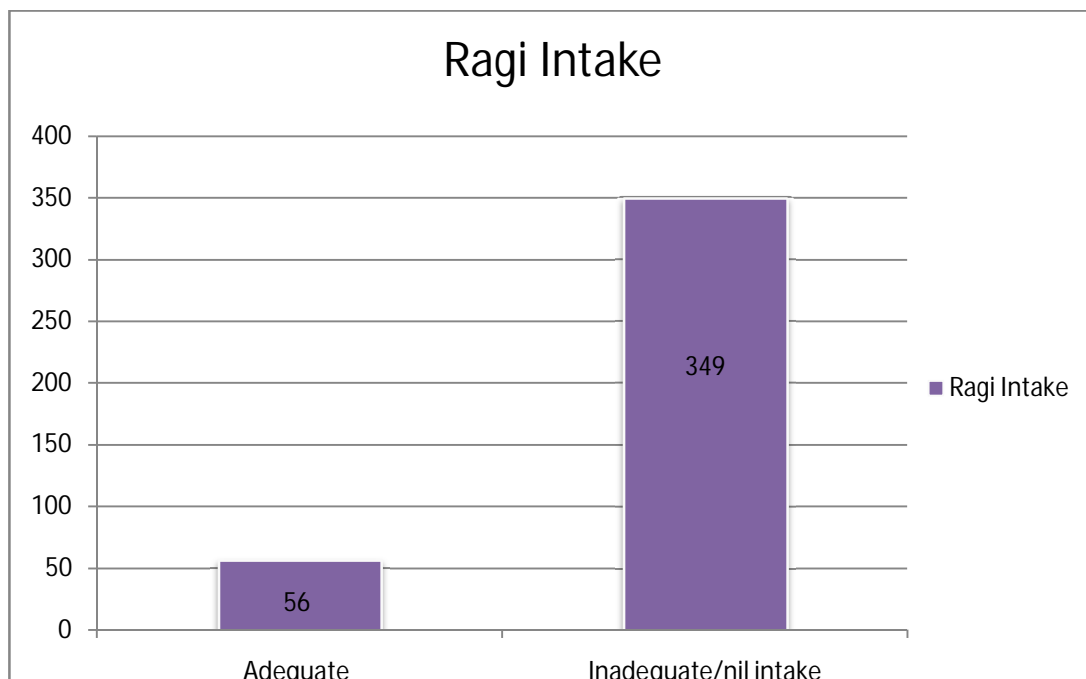


Figure 9: Prevalence of inadequate milk intake

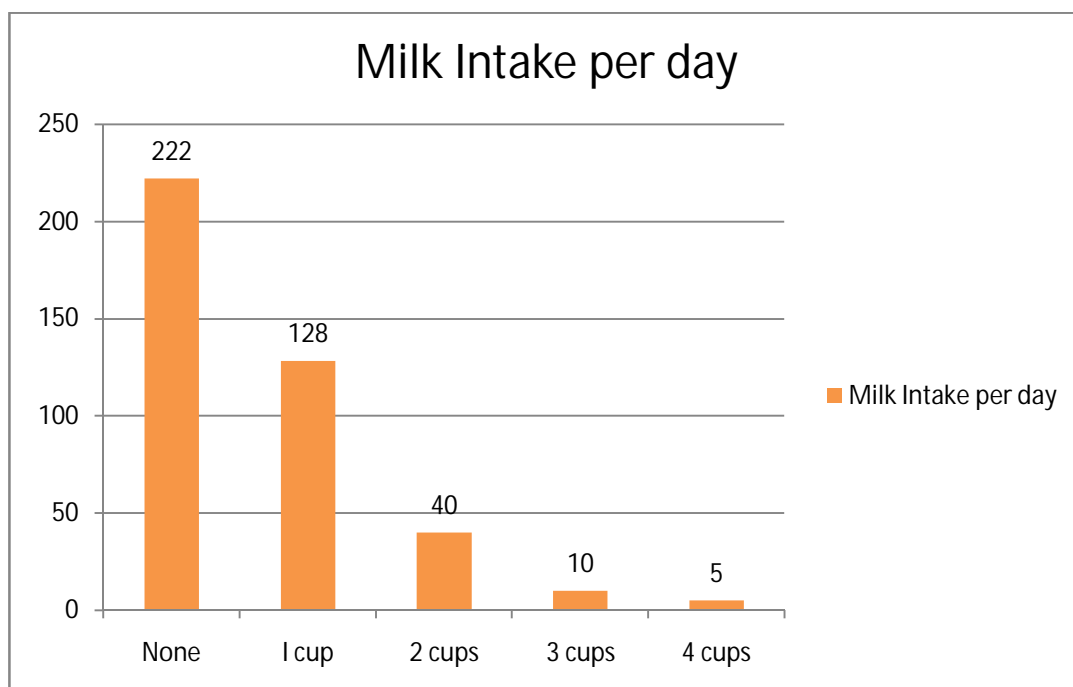


Figure 10: Intake of total caffeine units

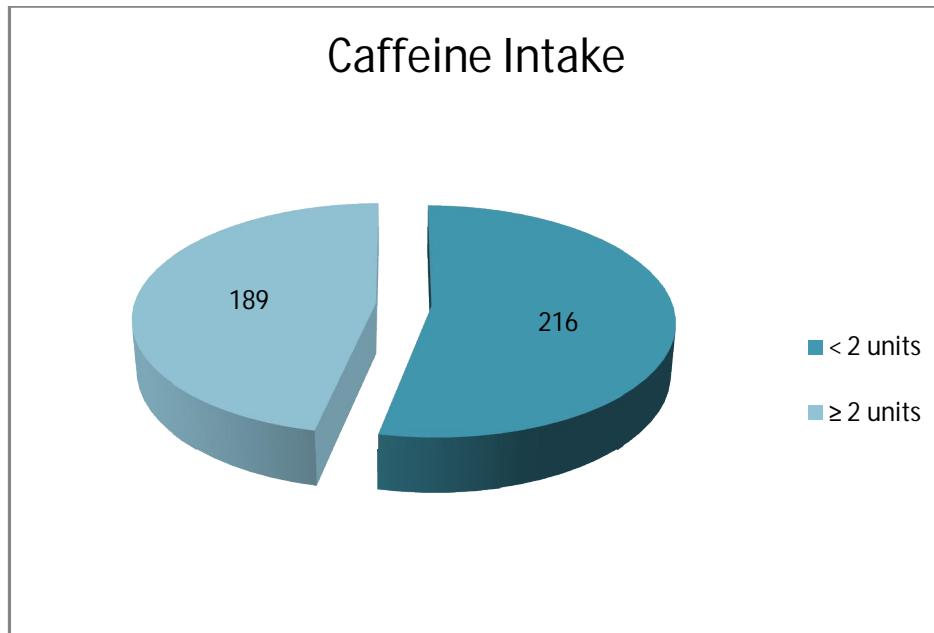
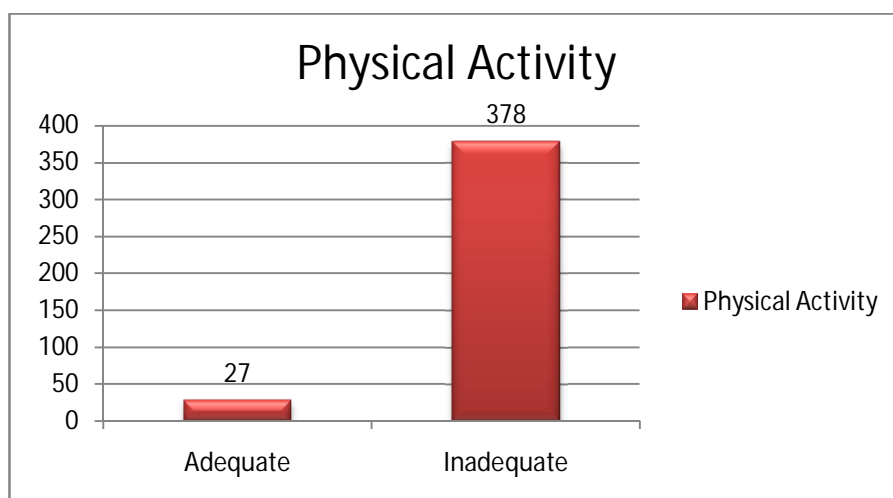


Table 8: Prevalence of other modifiable risk factors

Factor		Number	Percentage
Daily Sun Exposure	Yes	319	78.8
	No	86	21.2
Physical Activity	Yes	100	24.7
	No	305	75.3
BMI (kg/m ²)	< 18.5	28	6.9
	≥ 18.5	377	93.1

21.2% of the subjects are not exposed to the sun at least for 15 minutes each day. Only 24.7% of them engage in any sort of physical activity. Underweight individuals i.e. with BMI < 18.5 was 6.9%. The mean BMI of the male participants was 24.99 kg/m² with standard deviation 4.09 kg/m² and the mean BMI of the female participants was 25.21 kg/m² with standard deviation 5.31 kg/m².

Figure 11: Prevalence of inadequate physical activity



Among the 100 participants who are engaged in active physical work, only 27 participants are engaged in adequate amount of activity.

Table 9: Prevalence of tobacco and alcohol use among males

Factor		Number	Percentage
Current Smokers among males	Yes	29	21.2
	No	108	78.8
Ever Smoked among males	Yes	43	31.4
	No	94	68.6
Other forms of tobacco among males	Yes	12	8.8
	No	125	91.2
Alcohol intake among males	Yes	45	32.8
	No	92	67.2

The prevalence of current smoking among the males is 21.2% and 31.4% of the male subjects are currently smoking or have smoked before. 8.8% of the male subjects are using alternate forms of tobacco and alcohol is ingested by 32.8% of the males.

Only 1 female participant answered as a current smoker and 3 of them had ever smoked. 4 of the females consumed other forms of tobacco and 3 of them consumed alcohol.

Table 10: Prevalence of non modifiable risk factors

Factor		Number	Percentage
History of fracture	Yes	54	13.3
	No	351	13.3
Fracture in first degree relatives	Yes	73	18
	No	332	82

Among the non modifiable risk factors for low bone density, 13.3% of the subjects had self-reported history of fracture without heavy impact and 18% had relatives with history of easy fracturing.

Table 11: Prevalence of reproductive non modifiable risk factors among women

Factor		Number	Percentage
Late Menarche (> 16 years)	Yes	37	13.8
	No	231	86.2
Menopause or hysterectomy < 45 years	Yes	45	44.4
	No	36	55.6

Of the female participants, 81 women had attained menopause and among them 44.4% had attained menopause before the age of 45 years. Only 8 (3%) women among the study participants had used or were using hormones. None of the women who had attained menopause had taken any hormone replacement therapy (HRT).

Table 12: Prevalence of drug intake

Factor		Number	Percentage
Thyroxine	Yes	24	5.9
	No	381	94.1
Steroids	Yes	9	2.2
	No	396	97.8
Antacids	Yes	19	4.7
	No	386	95.3
Proton pump inhibitors	Yes	9	2.2
	No	396	97.8

5.9% and 2.2% of the subjects had history of chronic thyroxine or chronic steroid use, respectively. There was only one person taking anti-psychotics and anti epileptics each.

6.4 Low Bone Density and Socio-demographic Variables

Table 13: Socio-demographic distribution of low bone density

Variable		Normal Bone Density (%)	Low Bone Density (%)	Total	Chi square	df	P value
Age Groups (in years)	18-30	52 (56.5)	40 (43.5)	92	6.771	3	0.08
	31-45	72 (47.7)	79 (52.3)	151			
	46 - 60	54 (41.2)	77 (58.8)	131			
	> 60	11 (35.5)	20 (64.5)	31			
Sex	Males	66 (48.2)	71 (51.8)	137	0.189	1	0.664
	Females	123 (45.9)	145 (54.1)	268			
Religion	Hindus	150(47.9)	163 (52.1)	313	0.881	2	0.644
	Christians	29 (42.6)	39 (57.4)	68			
	Muslims	10 (41.7)	14 (58.3)	24			
Socio economic class	Class I and II	96 (45.9)	113 (54.1)	209	0.093	1	0.760
	Class III, IV and V	93 (47.4)	103 (52.6)	196			
Total		189 (46.7)	216 (53.3)	405			

There is an increasing trend noticed in the prevalence of low bone density. The difference between the age groups was not statistically significant. Females (54.1%) have higher prevalence than males (51.8%). The association between sex of the individual, religious groups and socio economic classes and presence of low bone density is not statistically significant.

6.5 Modifiable risk factors and socio-demographic variables

6.5.1 Fish intake:

Table 14: Age and sex wise distribution of fish intake

Variable		Fish intake		Total	Chi square	df	P value
		>1/week (%)	<1/week (%)				
Age Groups	18-30	40 (43.5)	52 (56.5)	92	11.125	3	0.011
	31-45	82 (54.3)	69 (45.7)	151			
	46-60	61 (46.6)	70 (53.4)	131			
	> 60	7 (22.6)	24 (77.4)	31			
Sex	Males	61 (44.5)	76 (55.5)	137	0.474	1	0.491
	Females	129 (48.1)	139 (51.9)	268			
Total		190 (46.9)	215 (53.1)	405			

Individuals more than 60 years (22.6%) show the least intake of fish followed by those between 18-30 years of age (43.5%). A statistically significant difference was seen between the age groups and fish intake ($p=0.011$). 55.5% of males and 51.9% of females are also not consuming fish at least on one day of the week. No statistical association was seen.

6.5.2 Ragi Intake:

Table 15: Age and sex wise distribution of ragi intake

Variable		Ragi intake		Total	Chi square	df	P value
		>1/week (%)	<1/week (%)				
Age Groups	18-30	9 (9.8)	83 (90.2)	92	4.278	3	0.233
	31-45	22 (14.6)	129(85.4)	151			
	46-60	23 (17.6)	108 (82.4)	131			
	> 60	2 (6.5)	29 (93.5)	31			
Sex	Males	15 (10.9)	122 (89.1)	137	1.439	1	0.230
	Females	41 (15.3)	227 (84.7)	268			
Total		56 (13.8)	349 (86.2)	405			

All age groups showed a consistently low intake of ragi. Only 10.9% of males and 15.3% of females consumed ragi on one or more days of a week. No statistical significance was seen between the age groups or gender based on ragi intake.

6.5.3 Milk intake:

Table 16: Age and sex wise distribution of milk intake

Variable		Milk intake		Total	Chi square	df	P value
		2 or more cups (%)	≤ 1 cups (%)				
Age Groups	18-30	17 (18.5)	75 (81.5)	92	9.049	3	0.029
	31-45	13 (8.6)	138 (91.4)	151			
	46-60	17 (13)	114 (87)	131			
	> 60	8 (25.8)	23 (74.2)	31			
Sex	Males	18 (13.1)	119 (86.9)	137	0.034	1	0.853
	Females	37 (13.8)	231 (86.2)	268			
Total		55 (13.6)	350 (86.4)	405			

Individuals in the older age group (>60 years) had higher milk intake (2 or more cups) than the other age groups. Percentage of milk intake among males and females was almost equal. Statistical significant difference was seen between the age groups.

6.5.4 Caffeine intake:

Table 17: Age and sex wise distribution of caffeine intake

Variable		Caffeine intake		Total	Chi square	df	P value
		< 2 caffeine units (%)	≥2 caffeine units (%)				
Age Groups	18-30	65 (70.7)	27 (29.3)	92	21.162	3	0.000
	31-45	81 (53.6)	70 (46.4)	151			
	46-60	52 (39.7)	79 (60.3)	131			
	> 60	18 (58.1)	13 (41.9)	31			
Sex	Males	62 (45.3)	75 (54.7)	137	5.428	1	0.020
	Females	154 (57.5)	114 (42.5)	268			
Total		216 (53.3)	189 (46.7)	405			

A statistically significant difference was noted between the age groups and sexes in the total caffeine units consumed.

6.5.5 Sun exposure:

Table 18: Age and sex wise distribution of sun exposure

Variable		Sun exposure		Total	Chi square	df	P value
		Yes (%)	No (%)				
Age Groups	18-30	64 (69.6)	28 (30.4)	92	16.858	3	0.001
	31-45	121 (80.1)	30 (19.9)	151			
	46-60	115 (87.8)	16 (12.2)	131			
	> 60	19 (61.3)	12 (38.7)	31			
Sex	Males	115 (83.9)	22 (16.1)	137	3.316	1	0.069
	Females	204 (76.1)	64 (23.9)	268			
Total		319 (78.8)	86 (21.2)	405			

Greater proportion of people in the 18-30 year age group (30.4%) and more than 60 year age group (38.7%) are not exposed to adequate sunlight with significant difference seen between the age groups in sunlight exposure. Greater number of females (23.9%) are unexposed to sun daily for at least 15 minutes.

6.5.6 Physical activity:

Table 19: Age and sex wise distribution of physical activity

Variable		Physical activity		Total	Chi square	df	P value
		Adequate (%)	Inadequate (%)				
Age Groups	18-30	6 (6.5)	86 (93.5)	92	8.091	3	0.044
	31-45	4 (2.6)	147 (97.4)	151			
	46-60	13 (9.9)	118 (90.1)	131			
	> 60	4 (12.9)	27 (87.1)	31			
Sex	Males	18 (13.1)	119 (86.9)	137	13.937	1	0.000
	Females	9 (3.4)	259 (96.6)	268			
Total		27 (6.7)	378 (93.3)	405			

Large proportions of people in all age groups are physically inactive with >60 years age group showing highest level of physical activity (12.9%). Females were more physically inactive (96.6%) compared to males (86.9%). There is a statistically significant difference seen between age groups and sexes for physical activity.

6.5.7 Body Mass Index:

Table 20: Age and sex wise distribution of low body mass index

Variable		Body mass index (BMI)		Total	Chi square	df	P value
		$\geq 18.5 \text{ kg/m}^2$ (%)	$< 18.5 \text{ kg/m}^2$ (%)				
Age Groups	18-30	75 (81.5)	17 (18.5)	92	26.96	3	0.000
	31-45	149 (98.7)	2 (1.3)	151			
	46-60	124 (94.7)	7 (5.3)	131			
	> 60	29 (93.5)	2 (6.5)	31			
Sex	Males	128 (93.4)	9 (6.6)	137	0.038	1	0.845
	Females	249 (92.9)	19 (7.1)	268			
Total		377 (93.1)	28 (6.9)	405			

There were more number of underweight people in the younger age group (18-30years) as compared to the rest of the age groups. This was significant statistically. There is an increased proportion of females who are underweight (7.1%) as compared to 6.6% of males.

Tobacco and Alcohol

As the number of women smoking, taking alcohol or other forms of tobacco was negligible, analysis of men alone were taken into account for statistical comparison between the age groups.

6.5.8 Smoking:

Table 21: Age wise distribution of smoking

Age Groups	Smoking		Total	Chi square value	P value
	No (%)	Yes (%)			
18-30	17 (81)	4 (19)	21	2.653 [*]	0.452
31-45	38 (71.7)	15 (28.3)	53		
46 -60	38 (82.6)	8 (17.4)	46		
>60	15 (88.2)	2 (11.8)	17		
Total	108 (78.8)	29 (21.2)	137		

^{*}Fisher's Exact Test

6.5.9 Tobacco usage:

Table 22: Age wise distribution of tobacco use

Age Groups	Tobacco use		Total	Chi square value	P value
	Yes (%)	No (%)			
18-30	21 (100)	0 (0)	21	3.636 [*]	0.275
31-45	46 (86.8)	7 (13.2)	53		
46-60	43 (93.5)	3 (6.5)	46		
>60	15 (88.2)	2 (11.8)	17		
Total	125 (91.2)	12 (8.8)	137		

^{*}Fisher's Exact Test

6.5.10 Alcohol intake:

Table 23: Age wise distribution of alcohol intake

Age Groups	Alcohol intake		Total	Chi square value (df=3)	P value
	No (%)	Yes (%)			
18-30	16 (76.2)	5 (23.5)	21	8.040	0.043
31-45	28 (52.8)	25 (47.2)	53		
46-60	35 (76.1)	11 (23.9)	46		
>60	13 (76.5)	4 (23.5)	17		
Total	92 (67.2)	45 (32.8)	137		

47.2% of males in the 31-45 years age group consume alcohol. A statistically significant difference is noted among the age groups on consumption of alcohol.

6.6 Association between Low bone density and risk factors

Table 24: Low bone density and modifiable dietary risk factors

Variables		Low bone density		Total	Odds Ratio (95% CI)	Chi-square value (df=1)	P value
		No	Yes				
		Number (%)	Number (%)				
Milk intake	≥2 cups	28 (50.9)	27 (49.1)	55	1.217 (0.689 – 2.150)	0.460	0.498
	≤ 1 cup	161 (46)	189 (54)	350			
Total caffeine units	< 2 units	106 (49.1)	110 (50.9)	216	1.231 (0.832 – 1.821)	1.078	0.299
	≥ 2units	83 (43.9)	106 (56.1)	189			
Ragi intake	Adequate	33 (58.9)	23 (41.1)	56	1.775 (1.001- 3.147)	3.926	0.048
	Inadequate	156 (44.7)	193 (55.3)	349			
Fish intake	Adequate	96 (50.5)	94 (49.5)	190	1.340 (0.905 - 1.983)	2.142	0.143
	Inadequate	93 (43.3)	122 (56.7)	215			
Total		189 (46.7)	216 (53.3)	405			

Among those with low bone density (216, 53.3%), lower intake of milk, ragi and fish is seen than those having normal bone density. Those consuming more than 2 caffeine units per day are greater among people with low bone density (56.1%) than those with normal bone density (43.9%).

Those not consuming ragi on at least one day of a week have 1.775 odds of low bone density than those who consume ragi on at least a single day of the week. Statistically significant difference is noticed among those consuming and not consuming ragi on at least a day a week.

Table 25: Low bone density and other modifiable risk factors

Variables		Low bone density		Total	Odds Ratio (95% CI)	Chi-square value (df=1)	P value
		No	Yes				
		Number (%)	Number (%)				
Sun exposure	Yes	153 (48)	166 (52)	319	1.280 (0.791 – 2.072)	1.013	0.314
	No	36 (41.9)	50 (58.1)	86			
Physical activity	Adequate	17 (63)	10 (37)	27	2.036 (0.909 – 4.563)	3.087	0.079
	Inadequate	172 (45.5)	206 (54.4)	378			
BMI (kg/m ²)	≥ 18.5	174 (46.2)	203 (53.8)	377	0.743 (0.344 – 1.604)	0.576	0.448
	< 18.5	15 (53.6)	13 (46.4)	28			
Current smoker	No	175 (46.7)	200 (53.3)	375	1 (0.475 – 2.107)	0.000	1.000
	Yes	14 (46.7)	16 (53.3)	30			
Ever smoked	No	170 (47.4)	189 (52.6)	359	1.278 (0.686 – 2.382)	0.600	0.439
	Yes	19 (41.30)	27 (58.7)	46			
Alcohol intake	No	166 (46.5)	191 (53.5)	357	0.945 (0.517 – 1.727)	0.034	0.853
	Yes	23 (47.9)	25 (52.1)	48			
Total		189 (46.7)	216 (53.3)	405			

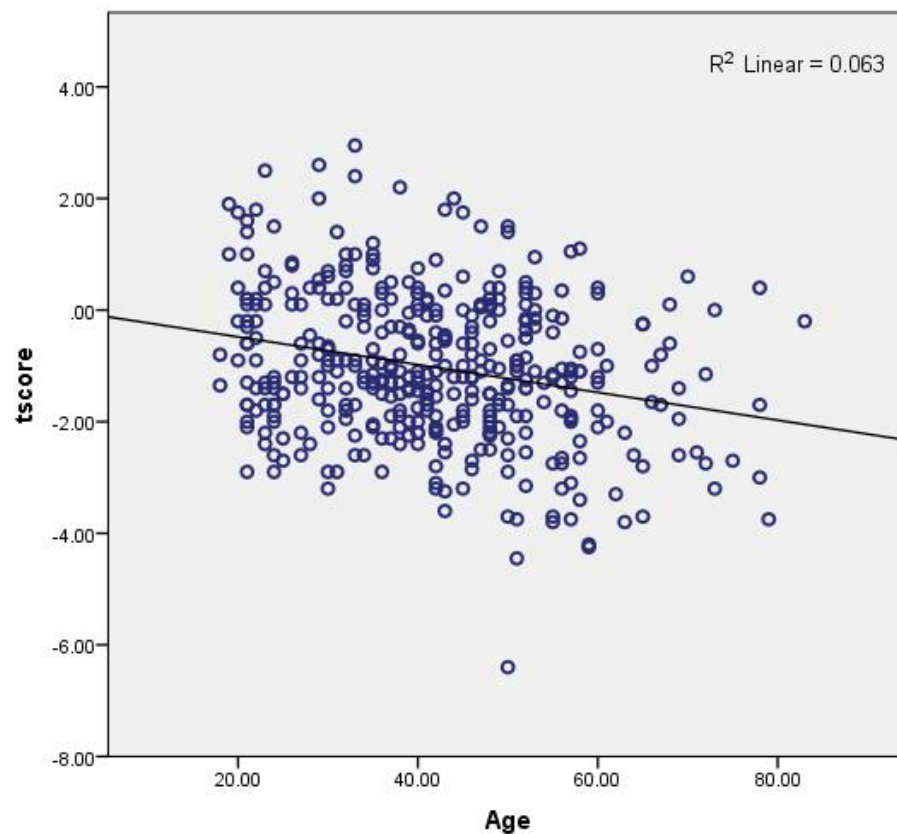
Higher prevalence of low bone density was seen among those who had inadequate sun exposure (58.1%) and inadequate physical activity (54.4%). No statistically significant difference was seen for low bone density in terms of sun exposure, physical activity, BMI, alcohol intake, current smoking or ever smoked status.

6.7 Correlation and regression

Table 26: Correlation between T score and age

		T score
Age	Correlation (r)	-0.250
	P-Value	0.000
	Number	405

Figure 12: Scatter Plot – Age versus T score

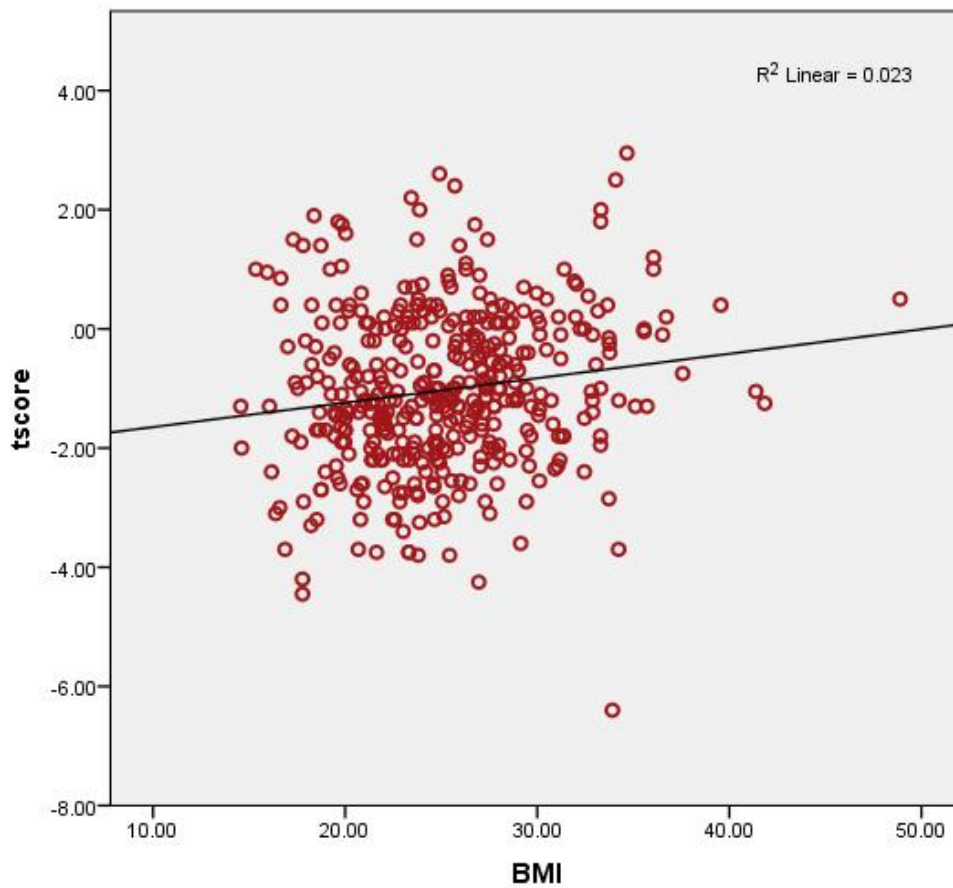


There is a negative correlation between age and the T score, i.e. decline in the T score as age advances and this is seen to be statistically significant ($r = -0.250$, $n = 405$, $p = 0.000$).

Table 27: Correlation between T score and BMI

		T score
BMI	Correlation	0.152
	P-Value	0.002
	Number	405

Figure 13: Scatter Plot – BMI versus T score



As the BMI increases an increase in the T score is seen. The positive correlation seen between the two variables is statistically significant ($r=0.152$, $n=405$, $p=0.002$).

Table 28: Multiple Logistic Regression Analysis

Variable		Beta	S.E. of Beta	P-Value	AdjustedOR	95% C.I. for EXP(B)	
						Lower	Upper
Age Group	18-30 years*						
	31- 45 years	0.383	0.271	0.158	1.467	0.862	2.497
	46 – 60 years	0.735	0.283	0.009	2.086	1.198	3.634
	> 60 years	0.960	0.443	0.030	2.612	1.096	6.227
Ragi intake	≥1/week*						
	<1/week	0.675	0.300	0.025	1.963	1.091	3.535
Physical activity	Adequate*						
	Inadequate	0.851	0.432	0.049	2.342	1.004	5.464

*Reference category

The multiple logistic regression shows that the factors causing low bone density after controlling for the effect of other variables are increasing age groups, inadequate ragi intake and inadequate physical activity. The individuals in the 46-60 year age group have a 2.086 higher odds of developing low bone density compared to those in the 18-30 year group and those above 60 years of age have 2.612 higher odds of developing low bone density. Both the odds are statistically significant.

Those not consuming ragi at all or inadequately have 1.963 times higher odds of developing low bone density than those consuming it on at least one day a week. This was significant at the 0.05 level ($p=0.025$).

Individuals who do not exercise adequately have a 2.343 times higher odds of developing low bone density compared to those who exercise adequately and this was statistically significant ($p=0.049$).

DISCUSSION

7. DISCUSSION

The current study is a community based study conducted to estimate the prevalence of low bone density, i.e. osteopenia and osteoporosis and the distribution of its various associated modifiable risk factors among the individuals aged 18 years and above in Nanganallur. The study carries significance for the public health threat and huge socio economic problem that osteoporosis and its consequent fractures carry and for the simple preventive measures that may be instituted at a young age for healthy bones in later age for the individuals and a sound backbone for the country's economy in the future.

The number of study participants was 405 with 137 males (33.8%) and 268 females (66.2%). The mean age of the male study participants was a slightly higher 45.2 years compared to 40.23 years in the females. The modified Kuppuswamy scale has been used to classify the socio economic status of the participants.

7.1 Prevalence of low bone density

The bone density of the participants was measured using the quantitative ultrasound technique which reported the result as a T score. This was interpreted by the investigator using the WHO guidelines on low bone density T scores. The prevalence of low bone density in the study population was 53.3% with 40% of osteopenia and 13.3% of osteoporosis (Table 6 & Figure 1). A study by Babu et al in north Kerala showed the prevalence of low

bone density to be 82.7% with 40.5% of osteopenia and 42.2% of osteoporosis.¹³ The high prevalence noted in the latter study could be due to the and the camp based approach that was used and the higher mean age (52 ± 12.8 years) of the participants compared to the current study (41.9 ± 13.4 years). A study conducted at Loni among the health care professionals between the ages of 21 – 61 years noted a prevalence of 31.06% of osteopenia and 28.03% of osteoporosis.¹⁵ The higher proportion of low bone density could be due to the selective picking of health care professionals and conducting a hospital based study.

The current study shows an increase in the proportion of osteoporotics (7.6%, 7.2%, 18.32%, 38.7%) with increasing age groups (18-30, 31-45, 46-60, >60years) and also a decline in the proportion having normal bone density (56.5%, 47.6%, 41.2%, 35.4%) (Figure 2). This is consistent with expected pattern of bone density and age.⁴⁸ The T scores also show a negative correlation with age (Table 26 & Figure 12) as seen in other studies.^{48, 49}

The current study shows an increased prevalence of low bone density among the females (54.1%) compared to the males (51.8%) but the proportion of osteoporotics is more among the males (14.6%) compared to the females (12.7%) (Figure 3). This could be due to the higher mean age of the male participants than the females. In a study by Prasad, 49% of the males 71% of the females had low bone density.¹⁵ The prevalence among males though somewhat similar among the males shows a wide variation among females. This may be due to the biased selection of individuals in the study. In Pande's

study 24.3% of the men and 29.9% of the women had low bone mass.¹⁶ The current study has employed QUS as a screening tool for low bone density whereas the latter has employed radiogrammetry which yields fewer false positive results.

The prevalence of osteopenia is slightly higher among the Muslim population (45.8%) and the prevalence of osteopenia is slightly higher among the Christians (16.2%) (Figure 4). The proportion of low bone density among the various socio economic classes does not seem to vary much except the 100% of the lower class people having osteoporosis but it has to be interpreted in the light of the observation that only 2 participants from that class were included in the study (Figure 5).

7.2 Prevalence of risk factors

The prevalence of each of the modifiable risk factor for low bone density was enumerated. From table 6, the consumption of dietary factors that contribute to low bone density can be seen. 23% of the population do not consume fish at all which is a major source of calcium and 67.7% do not consume any ragi which is also a readily available rich calcium source.⁷⁴ Milk is not consumed by 54.81% of the participants. Among the people consuming fish, ragi or milk, the proportion have them in adequate amounts is again far lesser (Figures 7, 8 & 9). Studies show that the South Indian diet of healthy individuals has far lesser calcium than the recommended dietary allowance.⁷⁵ The avoidance of these rich calcium sources predisposes these participants to a state of deficiency thereby leading to low bone density. Calcium

supplementation apart from the diet is essential for Indians who are naturally having calcium deficient diets. Only 25 (6.1%) of the participants are having adequate calcium supplementation (Figure 6).

60.2% and 62% of the participants are coffee and tea consumers respectively. Almost half of the participants (47%) consume more than 2 caffeine units a day (Table 7 & Figure 10) which negatively impacts bone density through its action on osteoblasts.⁴⁵

The daily sun exposure for at least 15 minutes was not present among 21.2% of the individuals. Studies show that Indians are lacking Vitamin D in spite of the abundant sunlight in the country.⁷⁶ The avoidance of the sun in this situation perpetuates Vitamin D deficiency leading to inadequate calcium absorption.

Only 24.7% of the participants engage in some sort of physical activity but only 6.66% satisfy the criteria of adequate physical activity. Insufficient exercise has been shown to be a major risk factor for low bone density and consequent fractures.⁴³

Underweight i.e. BMI $<18.5 \text{ kg/m}^2$ was seen among 6.9% of the participants. This has been documented to be a risk factor for developing low bone density and obesity has been found to be protective of low bone density in previous studies.³⁵ The mean BMI among the females in this study was also high in the overweight range (25.21 kg/m^2) which may have resulted in the higher distribution of low bone density in the males.

The prevalence of smoking currently among the male participants was 21.2% and 31.4% of males were wither currently smoking or had smoked previously (Table 9). 8.8% of the males were using alternative forms of tobacco and 32.8% consumed alcohol. Only a handful of females engaged in these practices in the current study. Almost one – fifth and one –third of the males are at risk of low bone density due to their smoking and alcoholic habits respectively.

The prevalence of some of the non modifiable risk factors were also elucidated in the study. This was done to identify those who were at risk of developing low bone density and fractures so that they could be educated on prevention of falls and fractures. 13.3% of the subjects reported at least one fragility fracture and 18% had a first degree relative who had a fragility fracture.

Among the women, 13.8% had attained menarche later than 16 years of age and of the 81 women who had attained menopause or hysterectomy, 44.4% had had it before the age of 45 years putting these women at greater risk of low bone density due to the loss of the protective effect of estrogen.

Small percentages of people were taking drugs such as thyroxine, steroids, antacids and proton pump inhibitors. These people need to be carefully monitored and be advised to use the drugs judiciously and only when necessary without using them for continuous prolonged periods. Anti epileptics and anti psychotics were only used by one subject each.

7.3 Low bone density and socio-demographic variables

Low Bone Density was not significantly associated with age, sex, religion or socio – economic class in the present study (Table 13). It is proven that increasing age and the female gender are distinctly associated with the development of low bone density.⁴⁷⁻⁵⁰ The current study has more number of younger females and smaller number of people in each of the age groups. This has possibly failed to demonstrate the association between the age groups, sexes and low bone density. However a significant negative correlation was seen between age and the T scores of the individuals ($r = -0.250$, $n = 405$, $p = 0.000$) (Table 26). This shows that with increasing age there is a decline in the T scores that are representative of the bone density values.

7.4 Modifiable risk factors and socio-demographic variables

The association of age and sex with the risk factors was studied using chi square tests. Fish intake was statistically significant ($p = 0.011$) between the different age groups with people in the > 60 year and 18-30 year age groups showing the least intake. No sex difference was noted in fish intake. No difference in ragi intake was noticed among the various age groups and the sexes. All age groups and both sexes are showing a consistently low consumption of such calcium rich diet.

Milk consumption patterns were significantly different among the various age groups ($p = 0.029$) but not between sexes. Elderly above 60 years had the highest percentage of adequate milk intake (25.8%) showing that

individuals of lower age groups are avoiding milk. Caffeine consumption was significant between the age groups ($p=0.000$) and sexes ($p=0.020$) showing highest consumption again among the 46-60 year age group and among the males.

Significant difference was noticed in the adequacy of sun exposure among the age groups ($p=0.001$) but not among the sexes. The elderly have the highest percentage of non exposure (38.7%) followed by the 18 -30 years group and hence reflects their lifestyle patterns of staying indoors. Adequate physical activity is dismally low among all age groups and both sexes but a significant difference is noted between the age groups ($p=0.044$) and sexes (0.000). Elderly are more active and the females are less active. The BMI shows a significant difference between age groups ($p=0.000$) as the youngest age group has a large proportion of individuals who are underweight (18.5%).

Statistically significant difference was observed between age groups (0.043) in consumption of alcohol among males but not in smoking or tobacco use.

7.5 Low bone density and modifiable risk factors

Significant association was seen between ragi intake and the presence of low bone density ($p=0.048$). No association was noticed between any of the other dietary factors like milk, caffeine or fish and low bone density. A study conducted at Japan showed that those who consumed more fish had higher bone densities than those who consumed none or less.⁷⁷ The current study

fails to demonstrate such an association probably because of the numerous other factors which influence the absorption of calcium from fish and also because of the genetic differences between the Japanese and the Indians. A study conducted in Sri Lanka, people with close genetic linkage to Indians also failed to show any difference between the diet and bone density of individuals.³⁶

Other modifiable risk factors like sun exposure, physical activity, smoking status, alcohol intake and BMI also did not show any statistically significant association with low bone density. A study by Lekamwasam shows more physically active people to have significantly higher bone densities but this association was not brought out by the current study.³⁶ It may have been possible to bring out such an association had the physical activity been classified quantitatively rather than as categorically. A study by Funakoshi showed no relation between lifestyle factors like drinking, smoking and exercise as in the present study.³⁷

7.6 Correlation and regression

Correlation between the age and the T scores of the individuals showed a negative correlation and this was extremely significant ($r = -0.250$, $n = 405$, $p = 0.000$) (Table 26 & Figure 12). This reflects that with increasing age, there is a decline in the T scores which is a proxy for the bone density values. This correlation is on par with the known trends of bone density and age.

The BMI and the T scores of individuals show a positive correlation that is extremely significant ($r= 0.152$, $n=405$, $p=0.002$). This shows that with an increase in the BMI, an increase in the T scores are also seen. This is consistent with the results seen in other studies.^{37,78}

A multiple logistic regression model was created and the variables age groups, ragi intake and physical activity were seen to be independent risk factors for the development of low bone density after controlling for the effect of other variables in the current study (Table 28). The 31-45 year age group did not show any significant difference in the odds of developing low bone density but the 46-60 year group and > 60 year group showed 2.086 and 2.612 times higher odds of developing low bone density compared to the 18-30 year group which was significant.

Inadequate ragi consumption puts individuals at 1.963 times higher odds of developing low bone density than those consuming it on at least one day a week. This was significant at the 0.05 level ($p=0.025$). Inadequate exercise has 2.343 times higher odds of leading to low bone density compared to those who get adequate exercise and this was statistically significant ($p=0.049$). The sex variable was also included in the model but it did not show any significance.

The current study shows a high prevalence of low bone density among all age groups and both sexes. The prevalence of low bone density associated modifiable risk factors are also very high among the study population especially among the younger age group which may hinder them attaining peak bone mass and predisposing them to low bone density in later life.

SUMMARY

8. SUMMARY

The current study was a cross sectional study carried out among 405 participants of age 18 years and more to assess the prevalence of low bone density and its associated modifiable risk factors in Nanganallur, an urban area of Chennai. This

The study was carried out using a pretested semi structured questionnaire and a quantitative ultrasound machine which measured the individual's bone mineral density and reported the result in terms of T scores which were interpreted using the WHO guidelines.

The key findings of the study were as follows:

- The prevalence of low bone density among the study population was 53.3% with 40% prevalence of osteopenia and 13.3% of osteoporosis.
- A rising trend of osteoporosis and declining trend of normal bone density was seen with advancing age groups though not significantly. No significant difference was seen in the distribution of low bone density by sex, religion or socio economic class.
- The prevalence of modifiable dietary risk factors was high among the study population. Fish, ragi and milk intake was inadequate among 53%, 86.17% and 46.41% of the study participants, respectively. Caffeine in excess of 2 units was consumed by 46.6% of the participants.

- Adequate sun exposure was absent in 21.2% of the study population, adequate exercise was lacking in a staggering 93.3% of the study population and 6.9% of them were underweight.
- Alcohol was consumed by almost one third of the male study population and smoking was practiced by approximately one fifth of them.
- There was significant difference noted between the various age groups in consumption of fish, milk and caffeine. The age groups also showed significant difference for sun exposure, adequate exercise and BMI. Among the male participants significant age group differences was noted for alcohol intake.
- Significant sex differences in the risk factors were noticed for caffeine intake with males having higher intake and for exercise with females showing lesser physical activity.
- Low bone density was significantly associated with inadequate calcium intake before adjustment.
- A significant negative correlation was seen between age and the T scores showing that with age, the bone density decreases.

- A significant positive correlation was seen between BMI and the T score reflecting that with increase in the BMI, increase in the T scores is seen.
- Multiple logistic regression showed ragi intake, exercise and age to be independent risk factors for low bone density after controlling for other variables but sex was not seen to be a risk factor in the current study.

The study therefore highlights the high prevalence of hitherto undetected low bone density and the importance of catching them young for modification of risk factors so as to enable individuals to attain their maximum possible bone mass, reduce rate of later life bone loss and incidence of fractures.

LIMITATIONS

9. LIMITATIONS

1. The present study has used quantitative ultrasound as the tool for identifying individuals with low bone density which has lower precision than DEXA, the ideal diagnostic tool. This may have lead to some misclassifications.
2. Certain risk factors like alcohol intake and smoking which have a dose effect on low bone density have not been quantified by the amount consumed and hence have restricted value in being predicted as a risk factor in the current study.
3. Dietary habits have been used as a proxy for assessing an individual's calcium status in place of blood tests.
4. The study carries the inherent limitations of cross sectional studies, thereby disabling the understanding of true temporal relationships between the risk factors and low bone density.
5. The study has covered a very selective area due to resource constraints and hence limits the generalisabilty to other urban areas.

RECOMMENDATIONS

10. RECOMMENDATIONS

Based on the findings of the current study, the following recommendations are being put forward.

1. Low bone density, considered to be a disease of the aged has been silently ravaging all ages and both sexes of the community with an alarming prevalence of 53.3% in the study. This calls for screening programmes for those at high risk on account of the huge social and economic burden it places on the community and country and the adverse impact on the quality of lives of the affected individuals.
2. As low bone density has many risk factors which are amenable to modification, emphasis must be laid on the lifestyle factors like diet, physical activity, smoking cessation and alcohol restriction.
3. Health education interventions for building up of high peak bone mass so as to avoid development of low bone density must be specifically targeted at the younger age groups as they have contributed to the major proportion of people with risk factors in the current study.
4. Further longitudinal studies need to be taken upon the younger individuals to assess the effect of these risk factors on bone density.

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ANNEXURES

ANNEXURE I

PATIENT INFORMATION SHEET - ENGLISH

Title of the dissertation:

A Descriptive Study on the Prevalence of Low Bone Density and Associated Modifiable Risk Factors among individuals aged 18 years and above in Nanganallur, an Urban area of Chennai in 2012.

Presence of Low Bone Density leads to fractures more easily. It has risk factors which if identified early can be modified.

This study is an attempt to identify the magnitude of low bone density and associated risk factors among those above 18 years of age in Nanganallur.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

PATIENT INFORMATION SHEET - TAMIL

ஆராய்ச்சி தகவல் தாள்

சென்னை உள்ள நங்கநல்லூரில் வசிக்கும் 18 வயதிற்கு மேற்பட்டவர்களிடம் குறைந்த எலும்பு வலு மற்றும் அதன் காரணங்கள் சம்பந்தமான ஆராய்ச்சி நடைபெற்று வருகின்றது.

எலும்பு வலு குறைவதனால் எலும்பு முறிவுகள் ஏற்படுவதற்கு வாய்ப்புகள் அதிகம். குறைந்த எலும்பு வலுக்கான காரணங்களை கண்டறிந்தால் அவற்றை மாற்றி அமைக்க முடியும்.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க விரும்புகிறேன். இந்த ஆராய்ச்சியில் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்புக்கு ஏற்படாது என்பதையும் தெரிவித்துக் கொள்கிறேன்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறேன்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின் வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறேன்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின்போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்துக் கொள்கிறேன்.

ANNEXURE II
INFORMED CONSENT FORM - ENGLISH

Title of the dissertation:

A Descriptive Study on the Prevalence of Low Bone Density and Associated Modifiable Risk Factors among individuals aged 18 years and above in Nanganallur, an Urban area of Chennai in 2012.

Name of the participant:

Participant ID:

Age :

- (1) I have been explained in detail about the study and its procedure. I confirm that I had completely understood the study and have had the opportunity to ask questions.
- (2) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.
- (3) I understand that the principal investigator, others working on the investigator's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However I understand that my identity will not be revealed in any information released to third parties or published.
- (4) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s).
- (5) I agree to take part in the above study.

Signature of investigator

Signature of participant

Date:

INFORMED CONSENT FORM - TAMIL

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு :

சென்னை உள்ள நங்கநல்லூரில் வசிக்கும் 18 வயதிற்கு மேற்பட்டவர்களிடம் குறைந்த எலும்பு வலு மற்றும் அதன் காரணங்கள் பற்றிய ஆய்வு.

பெயர் :

தேதி :

வயது :

பால் :

ஆராய்ச்சி சேர்க்கை எண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தைத் தெரிவிக்கிறேன்.

எனக்கு (Quantitative Ultrasound) குவென்டிடேட்டிவ் அல்ட்ரா சவுண்ட் என்ற கருவியின் மூலம் எலும்பு வலு பரிசோதனை செய்துக் கொள்ள சம்மதம்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்ப்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரின் தான் பங்கு பெறுகிறேன் மற்றும் நான் இந்த ஆராய்ச்சியிலிருந்து எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

நான் எலும்பு வலு சம்பந்தமான நோய்கள் குறித்த இந்த ஆராய்ச்சியின் விவரங்களைக் கொண்ட தகவல் தாளைப் பெற்றுக் கொண்டேன்.

நான் என்னுடைய சுயநினைவுடன் மற்றும் முழு சுதந்திரத்துடன் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள சம்மதிக்கிறேன்.

கையொப்பம்

ANNEXURE III
QUESTIONNAIRE - ENGLISH

ID No:

Descriptive Study on Prevalence of Low Bone Density and modifiable risk factors among individuals aged 18 years and above in Nanganallur, an urban area in Chennai in 2012

Socio-demographic particulars:

1. Name
2. Age years
3. Sex ☐ Male ☐ Female
4. Religion ☐ Hindu ☐ Christian ☐ Muslim
5. Address Door No:Street:
Area:
Pin code:
6. Education of Head of Family
 - a. ☐ Illiterate b. ☐ Primary School c. ☐ Middle School
 - d. ☐ Higher Secondary e. ☐ Diploma f. ☐ Graduate
 - g. ☐ Postgraduate
7. Occupation of Head of Family
 - a. ☐ Not working b. ☐ Unskilled c. ☐ Semi skilled
 - d. ☐ Skilled manual e. ☐ Skilled non manual
 - f. ☐ Semi professional g. ☐ Professional
8. Family Income: / month
9. Socio economic status:

History regarding osteoporosis:

10. Have you ever had a fracture? ☐ Yes ☐ No
11. Have any of your first degree relatives (parents/ siblings/ children) ever had a fracture? ☐ Yes ☐ No
12. How many cups of milk do you take in a day? _____
cups
13. (i) Do you drink coffee? ☐ Yes ☐ No
(ii) If yes, how many cups in a day? _____
cups
(iii) Do you drink tea? ☐ Yes ☐ No
(iv) If yes, how many cups in a day? _____
cups
14. (i) Do you regularly take calcium supplements? ☐ Yes ☐ No
(ii) How long have you been taking them?
(iii) How frequently do you take them?
15. (i) Do you consume ragi? ☐ Yes ☐ No
(ii) If yes, how frequently?
a. ☐ > 5 times/week b. ☐ <5times/Week
c. ☐ Once in two weeks d. ☐ once a month
16. (i) Do you consume fish? ☐ Yes ☐ No
(ii) If yes, how frequently?
a. ☐ > 5 times/week b. ☐ 1 - 5times/Week
c. ☐ Once in two weeks d. ☐ once a month

17. Are you exposed to the sun for at least 15 mins everyday?

☐ Yes ☐ No

18. i) Do you exercise?

☐ Yes ☐ No

ii) If yes, what kind of exercise?

a. ☐ Brisk walking b. ☐ Running / Jogging

c. ☐ Skipping d. ☐ Bicycling

e. ☐ Swimming f. ☐ Aerobics g. ☐ Lifting weights

iii) How often do you exercise?

a. ☐ < once a week b. ☐ 1 to 4 days a week c. ☐ > 5 days/week

iv) How long do you exercise?

a. ☐ < 15 min b. ☐ 15-30 min c. ☐ 30min to 1 hr d. ☐ > 1hr.

19. (i) Do you have any systemic medical disorders? ☐ Yes ☐ No

(ii) If yes, specify.....

20. Do you take any of the following drugs?

a. Steroids ☐ Yes ☐ No

b. Thyroxine ☐ Yes ☐ No

c. Proton pump inhibitors ☐ Yes ☐ No

d. Antacids ☐ Yes ☐ No

e. Anti epileptics ☐ Yes ☐ No

f. Anti psychotics ☐ Yes ☐ No

21. Do you smoke? ☐ Yes ☐ No

22. Have you ever smoked? ☐ Yes ☐ No

23. Do you use any tobacco

products? ☐ Yes ☐ No

24. Do you consume alcohol? ☐ Yes ☐ No

If female,

25. At what age did you attain menarche?

- a. ☐ <10 yrs b. ☐ 10-12yrs c. ☐ 13–15yrs d. ☐ 16yrs and above

26. (i) Have you attained menopause? ☐ Yes ☐ No

(ii) If yes, at what age? years

27. (i) Have you had your uterus and ovaries removed (Hysterectomy)?

☐ Yes ☐ No

(ii) If yes, at what age? years

28. Have you ever taken any hormones (estrogen / progesterone / combination)?

☐ Yes ☐ No

Examination

29. Height (cm):

30. Weight (kg):

31. T – score (QUS):

QUESTIONNAIRE - TAMIL

வினாபட்டி

- 1) பெயர் :
- 2) வயது : 3) பாலினம் :
- 4) மதம் : அ) இந்து ஆ) முஸ்லிம்
இ) கிறிஸ்துவம்
- 5) முகவரி :
- 6) குடும்பத்தலைவரின் படிப்பு : அ) படிப்பறியாது ஆ) 1st – 5th Std.
இ) 6th – 12th Std. ஈ) Degree / Diploma
உ) இவை அல்லாதது (Others)
- 7) குடும்பத்தலைவரின் வேலை (தொழில்) :
- 8) குடும்பத்தின் மாத வருமானம் என்ன? :
- 9) குடும்பத்தில் எத்தனை உறுப்பினர்கள் :
- 10) எலும்பு முறிவு எப்பொழுதாவது ஏற்பட்டுள்ளதா?
அ) ஆம் ஆ) இல்லை
- 11) உங்கள் பெற்றோர் அல்லது உடன்பிறப்புகள் அல்லது குழந்தைகளுக்கு
எப்பொழுதாவது எலும்பு முறிவு ஏற்பட்டுள்ளதா?
அ) ஆம் ஆ) இல்லை
- 12) ஒரு நாளில் எத்தனை கோப்பை பால் அருந்துவீர்கள்?
அ) 0 ஆ) 1 - 2 இ) 3 ஈ) > 3
- 13) 1) காபி அருந்துவீர்களா? அ) ஆம் ஆ) இல்லை
ஆம் என்றால் எத்தனை முறை.....
2) தேநீர் அருந்துவீர்களா? அ) ஆம் ஆ) இல்லை
ஆம் என்றால் எத்தனை முறை.....
- 14) கேல்சியம் மாத்திரை தொடர்ந்து உட்கொள்கிறீர்களா?
அ) ஆம் ஆ) இல்லை
- 15) போதுமான அளவு காற்றோட்டம் வீட்டில் உள்ளதா? : ஆம் இல்லை
1) ஆம் என்றால் எவ்வளவு நாட்களாக உட்கொள்கிறீர்கள்:
2) எத்தனை முறை உட்கொள்கிறீர்கள் :
- 16) கேழ்வரகு உண்பீர்களா?
அ) ஆம் ஆ) இல்லை

ஆம் என்றால் எத்தனை முறை உண்பீர்கள்?

- 1) வாரத்திற்கு 5 முறைக்கு மேல் 2) வாரத்திற்கு 5 முறைக்கு கீழ்
3) இரு வாரங்களில் ஒரு முறை 4) மாதத்திற்கு ஒரு முறை

17) மீன் உண்ணும் பழக்கம் உண்டா?

- அ) ஆம் ஆ) இல்லை

ஆம் என்றால் எத்தனை முறை உண்பீர்கள்?

- 1) வாரத்திற்கு 5 முறைக்கு மேல் 2) வாரத்திற்கு 5 முறைக்கு கீழ்
3) இரு வாரங்களில் ஒரு முறை 4) மாதத்திற்கு ஒரு முறை

18) வெளியிலில் தினமும் கால் மணி நேரமாவது இருப்பதுண்டா?

- அ) ஆம் ஆ) இல்லை

19) உடற்பயிற்சி செய்யும் பழக்கம் உண்டா?

- அ) ஆம் ஆ) இல்லை

1) ஆம் என்றால் எவ்வகையான உடற்பயிற்சி?

- 1) நடை பயிற்சி 2) ஓடுதல் 3) ஸ்கிப்பிங் 4) இரு சக்கரம் ஓட்டுதல்
5) நீச்சல் 6) அரோபிக்ஸ் 7) பல தூக்குதல்

2) எத்தனை முறை உடற்பயிற்சி செய்வீர்கள்?

- 1) வாரத்திற்கு ஒரு முறை 2) வாரத்தில் 1 - 4 நாட்கள்
3) வாரத்திற்கு 5 நாட்கள் அல்லது அதற்கு மேல்

3) எவ்வளவு நேரம் உடற்பயிற்சி செய்வீர்கள்?

- 1) < 15 நிமிடங்கள் 2) 15 - 30 நிமிடங்கள்
3) 30 நிமிடம் - 1 மணி நேரம் 4) 1 மணி நேரத்திற்கு மேல்

20) ஏதேனும் மருத்துவ கோளாறுகள் உண்டா?

- அ) ஆம் ஆ) இல்லை

1) ஆம் என்றால் என்ன

21) கீழ்வரும் மருந்துகள் ஏதேனும் உட்கொள்கிறீர்களா?

- அ) ஸ்டிராய்ட்ஸ் ஆம் இல்லை
ஆ) தைராக்சின் ஆம் இல்லை
இ) Proton Pump Inhibitors ஆம் இல்லை
ஈ) Antacid ஆம் இல்லை
உ) Anti epileptics ஆம் இல்லை
ஊ) Anti Psychotics ஆம் இல்லை

22) புகை பிடிக்கும் பழக்கம் உண்டா?

- அ) ஆம் ஆ) இல்லை

23) எப்பொழுதாவது புகை பிடித்ததுண்டா?

- அ) ஆம் ஆ) இல்லை

24) புகையிலை பொருட்கள் ஏதேனும் உபயோகிக்கும் பழக்கம் உண்டா?

- அ) ஆம்ஆ) இல்லை

- 25) மது அருந்தும் பழக்கம் உண்டா?
அ) ஆம் ஆ) இல்லை

பெண்கள் என்றால்

- 26) எந்த வயதில் வயதிற்கு வந்தீர்கள்?
அ) < 10 வருடம் ஆ) 10 - 12 வருடம்
இ) 13 - 15 வருடம் ஈ) 16 வயதிற்கு மேல்

- 27) மாதவிடாய் நின்று விட்டதா?
அ) ஆம் ஆ) இல்லை

ஆம் என்றால் எந்த வயதில் :

- 28) கர்ப்பை அல்லது கருப்பை அறுவை சிகிச்சை மூலம் அகற்றப்பட்டுள்ளதா?
அ) ஆம் ஆ) இல்லை

ஆம் என்றால் எந்த வயதில் :

- 29) ஏதேனும் ஹார்மோன் மாத்திரைகள் (Estrogen, Progesterone / Combined)
உட்கொண்டதுண்டா? அ) ஆம் ஆ) இல்லை

பரிசோதனைகள்

- 30) உயரம்
31) எடை
32) T – Score

ANNEXURE IV

MODIFIED KUPPUSWAMY'S SOCIO ECONOMIC SCALE

Education of head of the family	Score
Professional or Honours	7
Graduate or Post-Graduate	6
Intermediate or Post-High-School Diploma	5
High School Certificate	4
Middle School Certificate	3
Primary School or Literate	2
Illiterate	1
Occupation	
Professional	10
Semi-Professional	6
Skilled manual worker	5
Skilled non manual worker	4
Semi-skilled worker	3
Unskilled worker	2
Unemployed	1
Family Income per month (in Rs.)	
>30375	12
15188-30374	10
11361 – 15187	6
7594 – 11361	4
4556 – 7593	3
1521 – 4555	2
≤1520	1

Total Score	Class	Description
26-29	I	Upper class
16-25	II	Upper middle
11-15	III	Lower middle
5-10	IV	Upper lower
<5	V	Lower

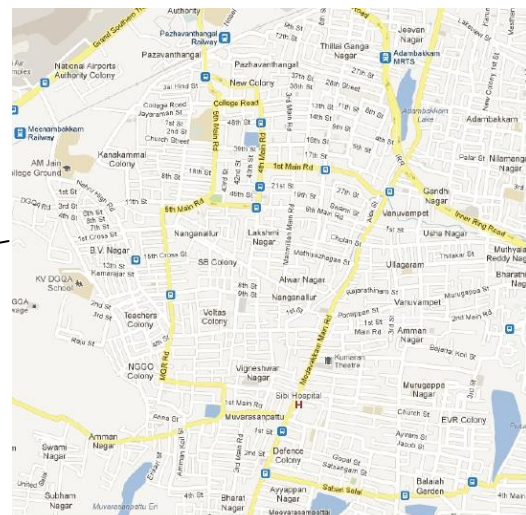
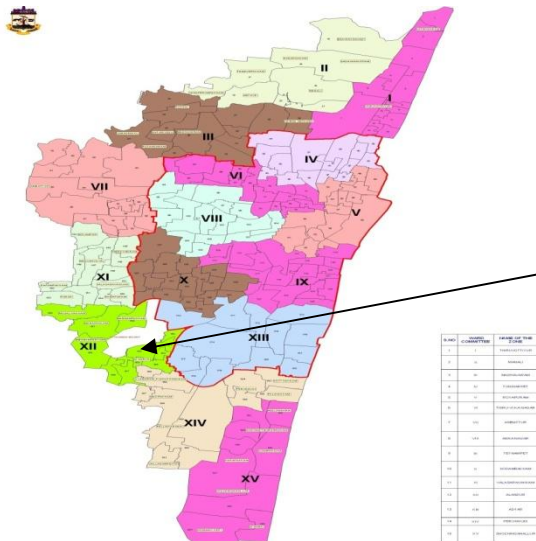
MAP OF TAMIL NADU

TAMIL NADU

Thiruvallur
Chennai
Vellore
Tiruvannamalai
Dharmapuri
Salem
Erode
The Nilgiris
Coimbatore
Namakkal
Tiruchirappalli
Perambalur
Ariyalur
Cuddalore
Nagapattinam
Karaikal
Thanjavur
Pudukkottai
Thiruvallur
Nagapattinam
Dindigul
Theni
Madurai
Sivaganga
Virudhunagar
Ramanathapuram
Tirunelveli
Kanniyakumari

P - Pondicherry

MAP OF NANGANALLUR



ANNEXURE VI

QUANTITATIVE ULTRASOUND MACHINE



ANNEXURE VII

LIST OF CLUSTERS IN NANGANALLUR

Cluster	population	cummulative frequency	cluster	population	cummulative frequency	cluster	population	cummulative frequency	cluster	population	cummulative frequency
1	135	135	76	88	11508	151	83	25541	226	210	35349
2	159	294	77	393	11901	152	513	26054	227	160	35509
3	183	477	78	239	12140	153	656	26710	228	182	35691
4	311	788	79	113	12253	154	86	26796	229	56	35747
5	82	870	80	90	12343	155	73	26869	230	34	35781
6	209	1079	81	373	12716	156	39	26908	231	137	35918
7	139	1218	82	258	12974	157	321	27229	232	118	36036
8	184	1402	83	86	13060	158	453	27682	233	117	36153
9	69	1471	84	47	13107	159	157	27839	234	263	36416
10	74	1545	85	69	13176	160	67	27906	235	271	36687
11	173	1718	86	92	13268	161	54	27960	236	409	37096
12	234	1952	87	318	13586	162	216	28176	237	85	37181
13	61	2013	88	639	14225	163	89	28265	238	47	37228
14	112	2125	89	77	14302	164	60	28325	239	97	37325
15	268	2393	90	453	14755	165	41	28366	240	63	37388
16	107	2500	91	63	14818	166	75	28441	241	71	37459
17	76	2576	92	51	14869	167	38	28479	242	32	37491
18	119	2695	93	38	14907	168	183	28662	243	321	37812
19	43	2738	94	138	15045	169	59	28721	244	72	37884
20	57	2795	95	212	15257	170	139	28860	245	61	37945
21	82	2877	96	233	15490	171	177	29037	246	45	37990
22	124	3001	97	128	15618	172	323	29360	247	317	38307
23	51	3052	98	230	15848	173	294	29654	248	69	38376
24	68	3120	99	83	15931	174	47	29701	249	141	38517
25	93	3213	100	139	16070	175	52	29753	250	48	38565
26	167	3380	101	202	16272	176	136	29889	251	43	38608
27	98	3478	102	46	16318	177	136	30025	252	103	38711
28	49	3527	103	52	16370	178	42	30067	253	38	38749
29	67	3594	104	264	16634	179	73	30140	254	84	38833
30	136	3730	105	149	16783	180	81	30221	255	57	38890
31	61	3791	106	363	17146	181	163	30384	256	26	38916
32	75	3866	107	84	17230	182	90	30474	257	128	39044
33	352	4218	108	117	17347	183	42	30516	258	107	39151
34	531	4749	109	57	17404	184	44	30560	259	211	39362
35	172	4921	110	62	17466	185	278	30838	260	73	39435
36	69	4990	111	136	17602	186	161	30999	261	95	39530

37	526	5516	112	75	17677	187	176	31175	262	81	39611
38	107	5623	113	124	17801	188	71	31246	263	432	40043
39	56	5679	114	381	18182	189	59	31305	264	194	40237
40	567	6246	115	217	18399	190	80	31385	265	27	40264
41	38	6284	116	76	18475	191	67	31452	266	187	40451
42	81	6365	117	91	18566	192	123	31575	267	254	40705
43	183	6548	118	465	19031	193	123	31698	268	82	40787
44	51	6599	119	268	19299	194	101	31799	269	64	40851
45	43	6642	120	174	19473	195	112	31911	270	57	40908
46	677	7319	121	54	19527	196	48	31959	271	31	40939
47	353	7672	122	33	19560	197	33	31992	272	433	41372
48	63	7735	123	283	19843	198	96	32088	273	129	41501
49	159	7894	124	452	20295	199	79	32167	274	115	41616
50	96	7990	125	162	20457	200	83	32250	275	43	41659
51	142	8132	126	76	20533	201	267	32517	276	139	41798
52	84	8216	127	391	20924	202	88	32605	277	72	41870
53	108	8324	128	215	21139	203	37	32642	278	256	42126
54	234	8558	129	48	21187	204	281	32923			
55	94	8652	130	32	21219	205	65	32988		1 st random number = 767	
56	42	8694	131	261	21480	206	104	33092		sampling interval = 1560	
57	89	8783	132	54	21534	207	72	33164	selected cluster are given in bold		
58	165	8948	133	576	22110	208	69	33233			
59	39	8987	134	773	22883	209	72	33305			
60	312	9299	135	319	23202	210	98	33403			
61	78	9377	136	68	23270	211	173	33576			
62	121	9498	137	281	23551	212	135	33711			
63	63	9561	138	137	23688	213	121	33832			
64	138	9699	139	126	23814	214	63	33895			
65	273	9972	140	283	24097	215	142	34037			
66	47	10019	141	64	24161	216	133	34170			
67	65	10084	142	29	24190	217	76	34246			
68	86	10170	143	56	24246	218	30	34276			
69	301	10471	144	132	24378	219	172	34448			
70	72	10543	145	197	24575	220	258	34706			
71	51	10594	146	162	24737	221	48	34754			
72	63	10657	147	234	24971	222	206	34960			
73	38	10695	148	412	25383	223	38	34998			
74	411	11106	149	32	25415	224	57	35055			
75	314	11420	150	43	25458	225	84	35139			

ANNEXURE VIII
KEY TO MASTER CHART

Variable	Label	Coding
ID No	Participant ID	1,2,etc
Age	Age of the participant	18,19,20..... years
Sex	Sex	0=Male 1=Female
Religion	Religion	0=Hindu 1=Christian 2=Muslim
Edn	Education of head of the family	1=illiterate 2=primary school 3=middle school 4=higher secondary 5=diploma 6=graduate 7=postgraduate
Occ	Occupation of head of the family	1=unemployed 2=unskilled 3=semi-skilled 4=skilled manual 5=skilled non-manual 6=semi-professional 7=professional
Income	Total income of the family	In Rupees
fractureH	Previous history of fracture	1=Yes 0=No
familyH	Family history of fracture	1=Yes 0=No
milk	Milk intake	0,1,2..... cups
coffee	Coffee consumption	1=Yes 0=No
coffeecups	Cups of coffee consumed	0,1,2..... cups
tea	Tea consumption	1=Yes 0=No
teacups	Cups of tea consumed	0,1,2..... cups
Calcium	Calcium supplementation	y=Yes n=No
Caldur	Duration of calcium supplementation	In months/years

Calfreq	Frequency of calcium supplementation	In days
Ragi	Ragi consumption	y=Yes n=No
ragifreq	Frequency of ragi consumption	a=>5times/week b=<5times/week c=once in 2weeks d=once a month
fish	Fish consumption	y=Yes n=No
fishfreq	Frequency of fish consumption	a=>5times/week b=<5times/week c=once in 2weeks d=once a month
exercise	Physical activity	0=Yes 1=No
exerkind	Type of exercise	a=brisk walking b=running/jogging c=skipping d=bicycling e=swimming f=aerobics g=lifting weights
exerfreq	Frequency of exercise	a=<1/week b=1-4days/week c=>5days/week
exerdur	Exercise duration	a=<15minutes b=15-30minutes c=30minutes to 1hour d=>1 hour
Sun	Exposure to sun	0=Yes 1=No
steroid	steroid intake	0=No 1=Yes
thyroxine	Thyroxine intake	0=No 1=Yes
PPI	Proton pump inhibitor use	0=No 1=Yes
Antacid	Antacid use	0=No 1=Yes
antiepi	Antiepileptic use	0=No 1=Yes

antipsy	Antipsychotic use	0=No 1=Yes
currmok	Currently smoking	0=No 1=Yes
eversmok	Ever smoked	0=No 1=Yes
tobacco	Tobacco use	0=No 1=Yes
alc	Alcohol use	0=No 1=Yes
menarche	Age of menarche	0= ≤16 years 1= >16years
menopause	Menopausal status	n=No y=Yes
menoage	Age at attaining menopause	In years
hys	Hysterectomy done	n=No y=Yes
hysage	Age at hysterectomy	In years
hormone	Hormone pills intake	n=No y=Yes
Ht	Height of the individual	In cm
Wt	Weight of the individual	In kg
tscore	T score of the individual	

ANNEXURE IX MASTER CHART

[illegible]

[illegible]

BRE-7	4	0	1	0	6	5	320 00	0	0	2	0	1	2	y	3 mon ths	alter rate day/s	y	c	n		1					0	0	0	0	0	0	0	0	0	0	0	y	42	n		n	16 3	7	0
BRE-8	4	0	0	0	6	5	230 00	1	0	0	1	4	1	2	n		y	b	y	b	1					0	0	0	0	0	0	0	0	0	0	0	1			n	16 8	9	0.4	
BRE-9	4	1	1	0	5	4	250 00	0	0	0	1	1	1	2	n		n		y	b	1					0	0	0	0	0	0	0	0	0	0	0	n		n	n	15 0	5	1.7	
BRE-10	5	9	1	0	3	3	120 00	0	0	0	1		0	2	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	y	41	n	n	n	16 0	6	1.2	
BRE-11	3	1	0	5	4		150 00	0	0	1	1	3	0		n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	0	n	n	n	16 0	8	0.3		
BRE-12	5	8	1	0	2	2	700 0	0	0	0	1	2	1	3	n		n		y	b	1				0	0	0	0	1	0	0	0	0	0	0	1	y	46	n	n	16 5	5	2.6	
BRE-13	3	1	0	6	1		200 00	0	0	1	0		0		n		n		y	b	1				0	0	0	0	0	0	0	0	0	0	0	n	n	n	15 0	6	1.4			
BRE-14	7	9	0	0	6	1	200 00	0	0	1	1	3	0		n		n		y	b	1				1	0	0	0	0	0	0	0	0	0	0		n	n	15 4	5	1.7			
BRE-15	6	2	1	0	5	5	150 00	0	0	0	0		0		n		n		y	b	1				0	0	1	0	0	0	0	0	0	0	0	0	n	n	n	15 4	5	0.8		
BRE-16	3	0	1	0	3	1	120 00	0	0	1	0		0		y	6 mon ths	ever yda y	n	n		1				1	0	0	0	0	0	0	0	0	0	0	0	n	n	n	15 5	4	0.6		
BRE-17	5	0	0	0	2	3	120 00	0	0	0	0	1	7	n		n		y	b	0	a	b	b	0	0	0	0	0	0	0	0	0	1	1	0	1			n	16 3	2	1.9		
BRE-18	3	2	0	0	6	5	150 00	0	0	0	0		0	4	n		n	n	n		1				1	0	0	0	0	0	0	0	0	0	0	1			n	16 8	7	1.9		
BRE-19	0	0	0	2	5		200 00	0	0	0	1	3	1	2	n		n		y	c	0	b	c	c	0	0	0	0	0	0	0	0	0	0	0			n	17 8	2	0.5			
DREN-1	2	0	0	5	3		180 00	0	0	2	1	1	1	1	n		y	b	y	b	1				0	0	0	0	0	0	0	0	0	0	0	0			n	16 2	7	0.9		
DREN-2	4	5	0	0	3	3	100 00	0	0	0	1	2	1	2	n		n		y	b	1				0	0	0	0	0	0	0	0	0	0	1	1			n	16 5	7	0.4		
NEN-1	6	0	1	0	3	1	120 00	0	0	2	1	1	1	1	y	15 year s	alter name day	y	b	n		1			0	0	0	1	1	0	0	0	0	0	0	y	52	y	52	n	15 6	8	1.2	
NEN-2	4	0	0	0	6	4	120 00	0	0	1	1	1	1	1	n		n	n			0	a	c	b	0	0	0	0	0	0	0	0	0	1	1	0	0			n	17 2	9	0	
NEN-3	5	0	2	4	5		400 00	0	0	0	1	1	1	2	y	10 day s	ever yda y	y	d	y	b	1				0	0	0	0	0	0	0	0	0	0	0			n	16 8	7	0.5		
NEN-4	4	1	0	6	4		250 00	0	0	0	1	1	1	5	n		y	d	n		1				0	0	0	0	0	0	0	0	0	0	0	n		n	14 3	5	1.5			
NEN-5	3	0	0	6	5		300 00	0	1	0	1	2	1	1	n		n		y	b	1				0	0	0	0	0	0	0	0	0	0	0			n	17 2	6	1.5			
NEN-6	3	1	0	7	1		200 00	0	1	2	0		0		n		y	b	y	b	0	d	a	b	0	0	0	0	0	0	0	0	0	0	0	n	n	n	15 0	7	2.9			
NEN-7	6	0	1	0	3	1	400 00	0	1	3	0		1	2	n		n		n		1				0	0	0	0	0	0	0	0	0	0	0	1	y	48	n	n	14 3	5	2.8	
NEN-8	5	0	0	7	1		800 00	1	0	0	1	1	1	1	n		n		y	0	b	b	d	0	0	0	0	0	0	0	0	0	0	0	0			n	16 5	6	3.2			
NEN-9	3	2	1	1	6	1	250 00	1	0	0	0		1	2	y	3 mon ths	dail y	y	b	y	b	1				0	0	0	0	0	0	0	0	0	0	n	n	n	15 0	8	0.4			
NEN-10	1	0	0	4	3		300 00	0	0	2	1	1	1	1	n		n		n		1				0	1	0	0	0	0	0	0	0	0	0			n	17 0	9	1.3			
NEN-11	5	1	0	0	6	5	500 00	1	0	1	1	1	1	1	n		y	d	y	b	1				0	0	0	0	0	0	0	0	0	0	0			n	17 2	6	1.7			
NEN-12	5	0	0	3	5		300 00	0	0	1	1	2	0		n		n		y	c	1				0	0	0	0	0	0	0	0	0	1	0	0			n	18 0	6	3.2		
NEN-13	3	0	1	2	1	2	500 0	0	0	0	1	1	1	3	n		n	n	n		1				0	0	0	0	0	0	0	0	0	0	0	n	n	n	16 8	5	1.4			
NEN-14	3	1	2	2	3		850 0	0	1	3	0		1	1	y	2 mon ths	ever yda y	y	b	y	b	1				1	0	0	0	0	0	0	0	0	0	y	45	n	n	15 0	2	1.2		
NEN-15	2	0	2	6	3		300 00	0	0	1	1	1	0		n		n		y	b	0	g	b	c	0	0	0	0	0	0	0	0	0	0	0			n	16 5	5	1.4			
NEN-16	1	0	4	3			600 0	0	1	1	1	1	1	2	y	1 year	ever yda y	n	y	b	0	f	c	a	0	0	0	0	0	0	0	0	0	0	y	48	n	n	14 5	0	2.1			
NEN-17	5	1	0	3	4		180 00	0	0	1	1	1	1	2	y	year s	ever yda y	y	a	y	b	0	a	c	b	1	0	0	1	0	0	0	0	0	0	y	43	y	43	n	14 3	5	2.5	
NEN-18	5	1	0	2	1		200 0	0	0	1	0		0		n		n		y	c	1				1	0	0	0	0	0	0	0	0	0	0	y	50	n	n	15 2	5	3.8		
NEN-19	0	1	0	6	3		100 00	0	0	0	1	2	1	1	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	n	n	n	15 5	7	0.9			
NEN-20	0	0	0	6	6		250 00	0	0	0	1	4	1	2	n		n		y	b	1				1	0	0	0	0	0	0	0	0	0	1	n		n	15 2	6	2.2			
NEN-21	4	0	0	6	5		200 00	0	0	1	1	1	1	1	n		n		y	b	0	a	c	b	0	0	0	0	0	0	0	0	0	0	0			n	17 0	8	1.3			
NEN-22	1	0	3	4			200 00	0	0	0	1	3	1	3	n		n		y	b	1				0	0	0	0	0	0	0	0	0	0	0	n	n	y	15 8	5	1.5			
NEN-23	4	1	0	2	3		400 0	0	0	0	1	1	1	1	n		y	a	y	b	1				0	0	0	0	0	0	0	0	0	0	y	40	n	n	15 5	7	0.6			
NEN-24	6	1	0	4	1		180 00	0	0	0	1	2	1	2	n		n		y	b	1				0	0	0	0	1	1	0	0	0	0	0	y	52	y	52	n	14 5	4	1.6	
NEN-25	1	0	4	1			250 00	0	0	0	1	2	0		n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	y	50	n	n	15 4	6	1.1		
NEN-26	1	1	0	4	5		300 00	0	0	1	0		0		y	3 mon ths	onc e a wee k	n		y	c	1				1	0	0	0	0	0	0	0	0	0	y	50	n	n	15 7	6	2.5		
NEN-27	3	3	1	0	4	5	300 00	0	0	0	0		1	3	n		n		n		1				0	0	0	0	0	0	0	0	0	0	0	n	n	n	15 4	6	0.1			
NEN-28	6	0	0	6	1		200 0	0	0	1	1	1	1	1	n		n		n		0	a	c	b	0	0	0	0	0	0	0	0	0	0			n	15 0	5	1.3				
NEN-29	5	1	1	0	4	4	200 00	0	0	1	1	1	1	1	n		y	c	n		1				0	0	0	0	0	0	0	0	0	0	y	48	n	n	15 9	6	0.9			
NEN-30	6	0	0	4	5		160 00	0	0	0	1	3	1	1	n		n		y	d	0	b	c	d	0	0	0	0	0	0	0	0	0	1	1	0			n	17 0	8	1.5		

1	0	7	4	140	0	0	0	1	1	0		n			y	d	y	d	1				0	0	0	0	1	0	0	0	0	0	0	0	n		n		n	16	8	-		
2	1	0	5	5	230	0	0	0	1	1	1	2	n		n		y	a	1				1	0	0	0	0	0	0	0	0	0	0	0	n		n		n	15	7	1.4		
3	0	0	2	3	800	0	0	0	1	3	1	1	n		n		y	b	1				0	0	0	0	0	0	0	0	1	1	1	1					15	7	1.8			
4	1	0	3	3	120	0	0	0	3	1	1	1	n		n		y	a	1				1	0	0	0	0	0	0	0	0	0	0	0	n		n		n	15	4	0.1		
5	1	0	6	4	150	0	0	1	0		1	4	n		n		n		0	a	a	a	1	0	0	0	0	0	0	0	0	0	0	0	n		n		n	16	3	1.1		
6	0	0	5	5	100	0	0	0	0		1	4	n		y	d	y	d	1				0	0	0	0	0	0	0	0	0	0	0	0					18	5	1.5			
7	0	0	4	5	500	1	0	0	1	2	0		n		n		y	d	0	a	b	b	0	0	0	0	0	0	0	0	0	1	0	0					16	5	-			
8	0	0	2	4	750	1	0	1	1	1	1	1	n		y	c	y	c	0	a	c	c	0	0	0	0	0	0	0	0	0	0	0	0					19	7	0.3			
9	0	0	7	6	400	0	0	0	1	1	1	2	n		y	d	y	d	0	a	a	c	0	0	0	0	0	1	0	0	0	0	0	0					17	1	-			
10	1	0	4	4	140	0	0	1	0		0		n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	0	n		n		n	16	4	1.3		
11	1	1	7	1	300	0	1	1	1	2	1	2	n		n		y	d	1				1	0	0	0	0	0	0	0	0	0	0	0	n		n		n	16	8	1.3		
12	1	1	5	4	170	1	1	0	1	3	1	3	n		n		n		1				0	0	0	0	0	0	0	0	0	0	0	0	n		n		n	19	0	-2		
13	1	0	4	4	800	0	0	1	0		1	1	n		n		y	d	1				1	0	0	0	0	0	0	0	0	0	0	0	n		n		n	16	1	-		
14	1	1	7	5	400	0	0	1	1	1	1	1	n		n		y	d	0	b	c	d	0	1	0	0	0	0	0	0	0	0	1	0	1	0	n		n		n	16	3	1.4
15	0	0	7	6	180	0	0	0	1	2	0		n		n		y	c	1				1	0	0	0	0	0	0	0	0	0	0	0					17	0	1.8			
16	0	1	3	3	100	0	0	0	1	1	1	1	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	0					16	7	2.6			
17	1	0	3	3	100	0	0	0	0		1	2	n		n		y	a	1				0	0	0	0	0	0	0	0	0	0	0	0	y	35	n		n	16	8	-1		
18	1	0	1	2	700	0	0	0	0		1	3	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	0	n		n		n	15	5	0.9		
19	1	1	5	4	160	0	0	0	0		1	1	n		y	b	y	c	1				1	0	0	0	0	0	0	0	0	0	0	0	n		n		n	14	8	3.1		
20	0	0	6	1	120	0	0	1	1	2	1	1	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	0					16	8	0.8			
21	1	0	7	5	400	0	0	0	1		0		n		n		n		1				0	0	0	0	0	0	0	0	0	0	0	0	n		n		n	15	5	0.1		
22	0	2	7	3	150	1	1	0	1	2	1	2	n		y	c	y	c	1				0	0	0	0	0	0	0	0	0	1	1	0	0					17	3	2.6		
23	0	0	5	4	200	0	0	1	1	3	0		n		n		n		1				1	0	0	0	0	0	0	0	0	0	1	1	0	1					17	1	-1	
24	0	0	4	4	150	0	0	1	0		1	3	n		n		n		1				1	0	0	0	0	0	0	0	0	0	1	1	0	1					16	9	1.9	
25	0	0	5	4	100	1	0	2	1	4	0		n		n		y	c	0	b	a	a	0	0	0	0	0	0	0	0	0	0	1	1	0	0					17	0	2.2	
26	0	0	4	1	100	0	0	1	1	2	1	2	n		n		n		0	a	b	b	0	0	0	0	0	0	0	0	0	0	1	0	0					16	5	0.1		
27	0	0	4	1	250	0	0	0	1	3	1	1	n		n		n		1	a	a	b	0	0	0	0	0	0	0	0	0	0	0	0	0					16	9	0.7		
28	0	0	2	4	700	0	0	0	1	2	1	1	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	1	1	0	0					16	5	-2	
29	1	0	4	4	140	0	0	0	0		1	4	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	0	y	47	n		n	15	2	2.6		
30	1	0	1	1	200	1	0	0	1	3	0		n		y	c	y	b	1				0	0	0	0	0	0	0	0	0	0	0	1	y	40	n		n	15	9	-		
31	1	0	2	2	500	0	0	0	1	3	1	2	n		n		y	a	1				0	0	0	0	0	0	0	0	0	0	0	0	y	47	n		n	14	9	0.9		
32	0	0	6	6	600	0	0	1	1	2	1	1	n		n		n		0	a	b	c	1	0	0	0	0	0	0	0	0	0	0	0	0					16	5	-		
33	0	0	6	6	200	0	0	2	1	1	1	1	n		y	c	n		1				0	0	0	0	0	0	0	0	0	0	0	0					16	6	1.4			
34	0	0	4	4	200	0	0	0	1	2	0		n		n		n		1				0	0	0	0	0	0	0	0	0	0	0	0	0					16	4	-1		
35	1	0	3	2	150	1	0	0	0		1	4	n		y	d	y	b	1				0	0	0	0	0	0	0	0	0	0	0	0	n		n		n	15	8	1.2		
36	0	0	6	5	180	0	1	1	1	1	1	2	n		n		y	d	1				0	0	0	0	0	0	0	0	0	0	0	0	0					18	0	1.9		
37	1	2	1	4	600	0	0	0	0		1	2	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	0	n		n		n	15	2	0.2		
38	1	0	7	1	500	1	0	2	0		1	2	n		n		n		0	d	c	b	1	0	0	0	0	0	0	0	0	0	0	0	n		n		n	14	4	1.8		
39	1	0	4	6	500	0	1	1	0		1	2	y	2 mon this	every yda 7	y	a	y	c	0	a	b	b	0	0	1	0	0	0	0	0	0	0	0	n		n		n	15	9	2.9		
40	1	1	5	4	300	0	0	0	1	2	0		n		y	d	y	d	1				0	0	0	0	0	0	0	0	0	0	0	0	y	45	n		n	15	0	3.4		
41	1	1	5	4	300	1	0	0	0		0		n		y	d	y	c	0	a	b	a	0	0	0	0	0	0	0	0	0	0	0	1	n		n		n	15	5	3.6		
42	1	1	2	3	700	0	0	0	0		1	2	n		y	a	y	b	1				0	0	0	0	0	0	0	0	0	0	0	0	1	y	44	n		n	15	1	1.5	
43	1	1	4	2	900	0	0	0	1	3	0		n		n		y	b	1				1	0	0	0	0	0	0	0	0	0	0	0	y	40	n		n	14	0	2.3		
44	1	0	2	2	900	0	0	0	0		1	3	n		n		y	c	1				0	0	0	0	0	0	0	0	0	0	0	0	y	42	n		n	15	0	-2		
45	1	0	4	4	200	0	0	0	1	1	2		n		n		y	d	1				1	0	1	0	0	0	0	0	0	0	0	0	1	n		n		n	18	0	1.9	

[illegible]

ANNEXURE X

ETHICAL COMMITTEE CLEARANCE CERTIFICATE

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301

Fax : 044 25363970

CERTIFICATE OF APPROVAL

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "A Descriptive study on the prevalence of Low Bone Density and Associated Modifiable risk Factors among individuals aged 18 years and above Nanganallur, an Urban area of Chennai in 2012" No.24072012.


The following members of Ethics Committee were present in the meeting held on 24.07.2012 conducted at Madras Medical College, Chennai -3.

- | | |
|--|---------------------|
| 1. Dr. S.K. Rajan. M.D.,FRCP.,DSc | -- Chairperson |
| 2. Prof. Pregna B. Dolia MD | -- Member Secretary |
| Vice Prinicipal, Madras Medical College, Chennai-3 | |
| Director , Inst. of Biochemistry, MMC, Ch-3 | |
| 3. Prof. Kalaiselvi MD | -- Member |
| Prof of Pharmacology ,MMC, Ch-3 | |
| 4. Prof. C. Rajendiran, MD | -- Member |
| Director , Inst. of Internal Medicine, MMC, Ch-3 | |
| 5. Prof. MD Ali M.D., D.M., | -- Member |
| Prof & HOD, Dept. of MGE, MMC, Ch-3 | |
| 6. Prof. S. Deivanayagam MS | -- Member |
| Prof of Surgery, MMC, Ch-3 | |
| 7. Thiru. S. Govindsamy, BABL | -- Lawyer |
| 8. Tmt. Arnold Soulina MA MSW | -- Social Scientist |

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.


Member Secretary, Ethics Committee

ANNEXURE XI

PLAGIARISM REPORT

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A DESCRIPTIVE STUDY ON THE PREVALENCE OF LOW BONE DENSITY AND ASSOCIATED MODIFIABLE RISK FACTORS AMONG INDIVIDUALS AGED 18 YEARS AND ABOVE IN NANGANALLUR, AN URBAN AREA OF CHENNAI IN 2012

32
Dissertation submitted to

THE TAMILNADU DR. MGR MEDICAL UNIVERSITY

In partial fulfillment of the requirements for the degree of

34
M.D. BRANCH XV

COMMUNITY MEDICINE

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